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EXTERNAL NASAL DEFORMITIES AND METHODS USED IN THEIR REPAIR

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The nose is such a conspicuous feature that its deformities and their corrections naturally arouse an appreciable general interest. Recently, however, that interest has been unduly intensified by ill advised articles on plastic surgery in some lay magazines and in the Sunday supplements of some metropolitan newspapers. Thus the public has become "nose conscious," and I fear that in too many instances emphasis has been placed on the repair of insignificant deformities in hypersensitive, introspective persons. I refer especially to minor abnormalities in the contour of the alar cartilages which, after operative procedures by insufficiently skilled surgeons, have been converted not infrequently into actual deformities of varying degrees, thus giving such patients ample justification for remaining in the "dissatisfied throng." This situation needs to be combated more efficiently by ethical plastic and reconstructive surgeons.

Real nasal deformities, however, are numerous; the problems encountered in their correction are intensely interesting, and in the hands of properly trained reconstructive surgeons, the functional as well as the cosmetic results are usually gratifying, both to the patient and to the surgeon.

The methods preferred for correction of the nasal deformities associated with the various types and degrees of cleft lip and cleft palate have been considered in detail in several of our previous papers, and thus will be omitted here.

Pronounced congenital deficiencies of the nasal bones and the frontonasal processes of the maxillas are preferably corrected at 17 to 20 years of age by autogenous rib cartilage transplants (fig. 1). If correction is attempted in childhood, the cartilage of course does not increase in

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size, and consequently as maturity is approached there is an increasing disproportion between the size of the transplanted cartilage and the size of the developing face. If, in some rare cases, early correction is deemed advisable because of marked introspection or other psychologic reason,

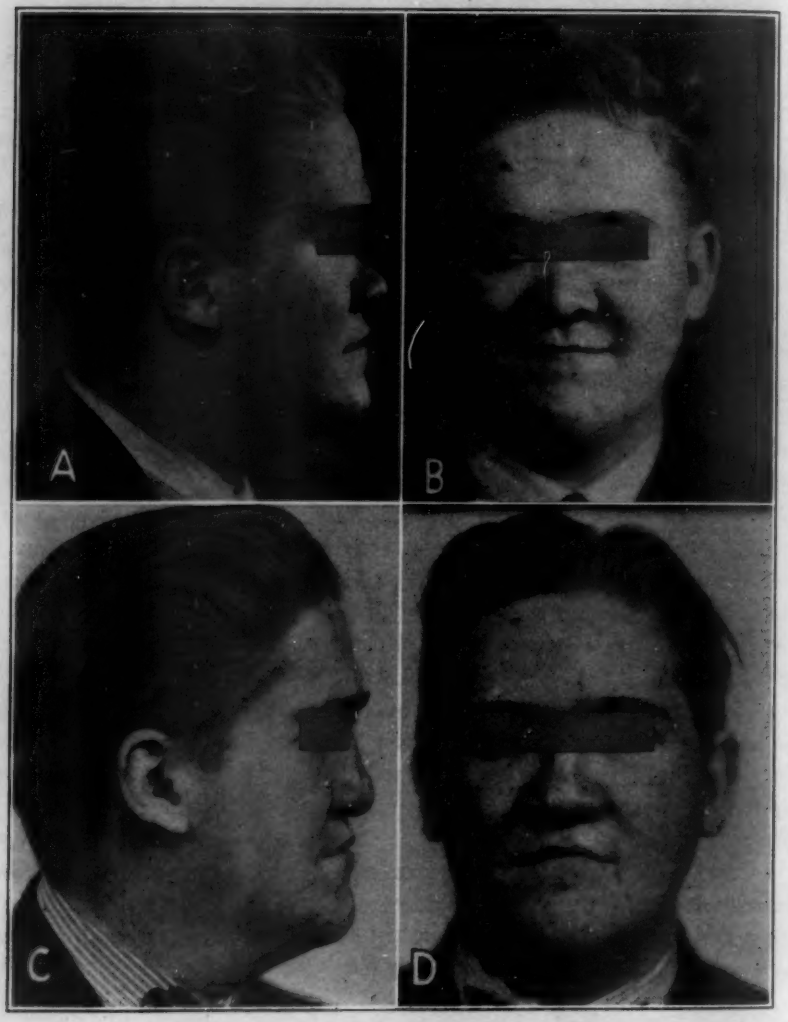

Fig. 1 (L. J., aged 19).—*A* and *B*, marked nonsyphilitic congenital deficiency of the nasal bones and frontonasal processes of the maxillas.

C and *D*, appearance three months after repair by a rib cartilage transplant.

the patients and the parents should be told that a second operation will probably be necessary during late adolescence.

For several months preceding operation, the depressed nasal area and the surrounding tissues should be firmly massaged twice daily to secure better circulation and sufficient looseness of the skin to avoid undue ten-

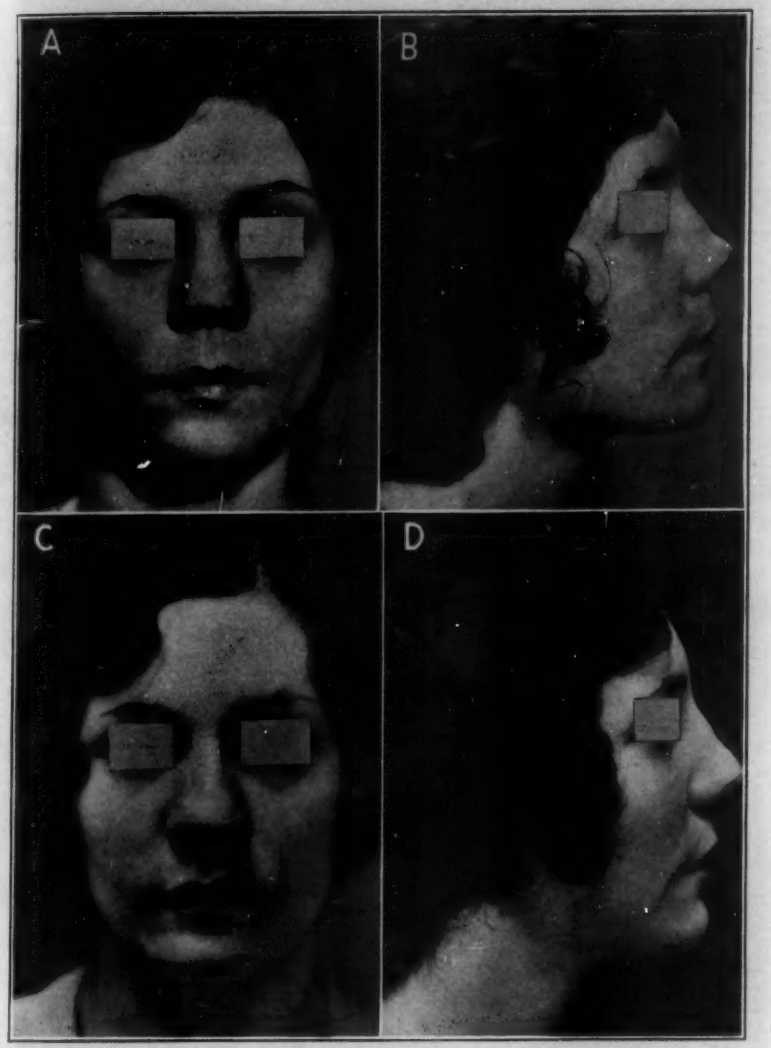

Fig. 2 (H. L., aged 19).—*A* and *B*, depression and shortening of the dorsum of the nose due to destruction of septal cartilage by an infected septal hematoma in childhood.

C and *D*, appearance six months after repair by a rib cartilage transplant to the dorsum of the nose and a small supporting cartilage transplant into the columella.

sion over the dorsum of the nose when a cartilage transplant of adequate size is placed in position. The use of curved costal cartilage chisels of

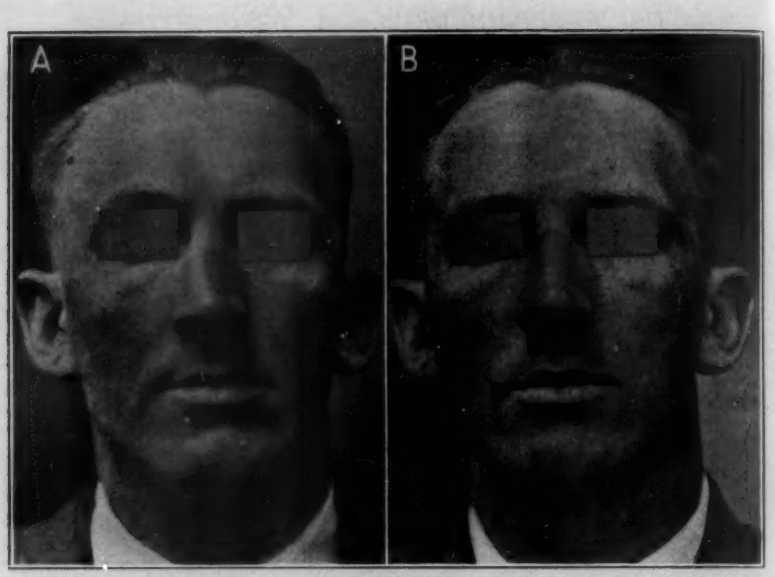

Fig. 3 (W. G., aged 26).—*A*, lateral deviation of nose from an old football injury.

B, appearance sixteen days after submucous resection of the obstructing portion of the septum, division of the frontonasal processes of the maxillas at the maxillary attachments and placing of structures in their normal position.

Fig. 4 (F. G., aged 21).—*A*, excess convexity of the dorsum and elongated, drooping tip of the nose.

B, appearance six months after removal of excess convexity of the dorsum, shortening and elevating the nasal tip.

proper sizes greatly facilitates obtaining the cartilage, and placing the smooth convex surface upward helps to insure a well formed dorsum nasi. The cartilage is preferably inserted through a columellar incision.

Nasal depressions from destruction of the septal cartilage by infected septal hematomas in childhood (fig. 2), those from too extensive sub-

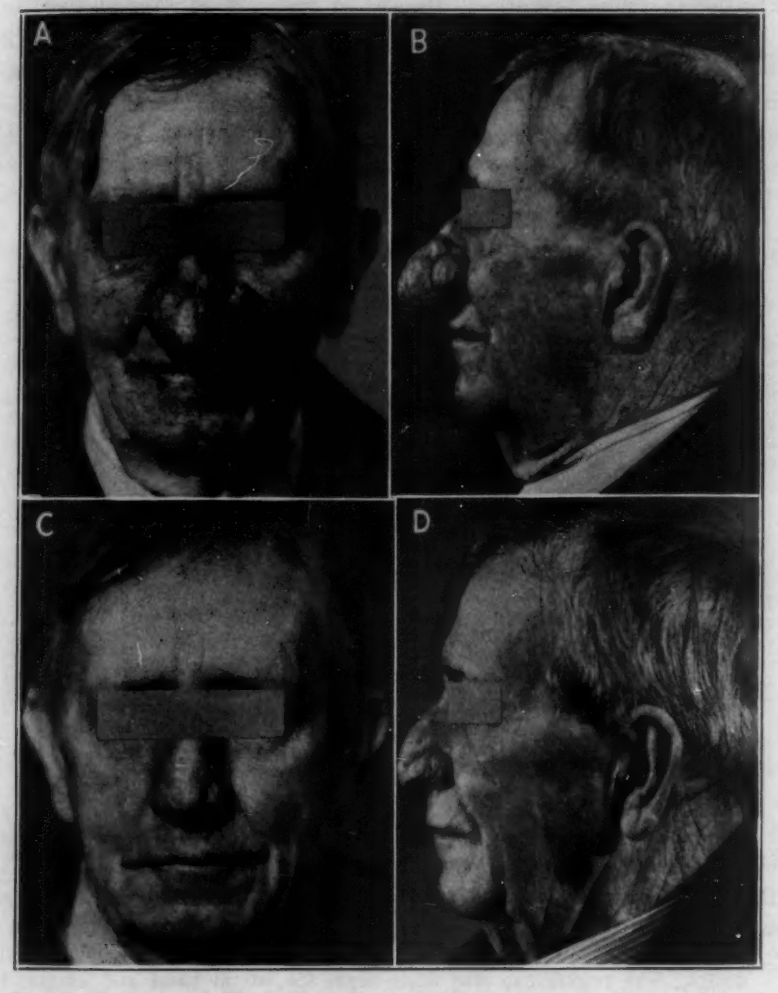

Fig. 5 (B. B., aged 68).—*A* and *B*, rhinophyma, of twenty-six years' development.

C and *D*, appearance two months after multiple elliptic excisions, made in two operations, one month apart.

mucous resections of the nasal septum and those from severe trauma are rather similarly corrected, but are more apt to require a supporting cartilaginous post in the columella, extending from the under surface of the anterior end of the dorsum transplant to the intermaxillary spine.

In some syphilitic depressions very large perforations of the nasal septum may interfere with the successful insertion of as large a piece of cartilage as is desired to correct the deformity adequately or to allow insertion of proper support for the anterior end of the cartilage transplant. In such instances one must necessarily be content with partial corrections.

Lateral deviations of the nose without depressions (fig. 3) are readily brought back to midline by partially dividing the frontonasal processes of the maxillas near their attachments to the bodies of the maxillas, either with a saw or with a thin narrow chisel, and fracturing the remaining

Fig. 6 (J. J. C., aged 38).—*A*, photograph showing base of the right ala excised by the family physician seven years previously, with a suspected malignant growth in the persisting fissure. (Excised tissue was not examined microscopically.)

B, appearance two months after repair by use of two single pedicle flaps. One flap was taken from just above the mustache line, left attached at the old base of the ala, rotated medially and sutured in position for the epithelial lining of the new nostril. The second flap was taken from the lateral nasal and infraorbital areas, rotated by approximately a one-fourth twist, bringing the raw surface in accurate contact with the raw surface of the first flap, and sutured at the margin of the new ala. Skin surrounding areas from which the flaps were taken was undercut and the edges advanced and sutured to cover the surfaces immediately.

attachments by digital pressure or by means of carefully placed mallets, in which latter case the skin must be adequately protected to avoid undue trauma. In many cases submucous resection of the deflected portion of

the septum is not only beneficial in giving a better respiratory area, but advantageous in preventing any spring or back pressure in the septum from tending to force the nasal structures back to the deviated position. In those cases in which there is a slight depression between the lower ends of the nasal bones and the nasal tip, the cartilage removed submucously should be trimmed to proper length and width and used to correct or to minimize the deformity by inserting it into the depth of the depression through an incision just within the superior margin of the anterior naris. If the depression is sufficiently great to require two thicknesses of cartilage, the pieces should be trimmed to the desired size and then fastened together by a mattress suture of fine catgut. This facilitates keeping the grafts in the proper position after insertion.

Fig. 7 (F. M., aged 75).—*A*, adenoid cystic epithelioma (low grade malignancy), of over twelve years' development. The inner surface of the nose was not involved. *B*, appearance nine weeks after rather wide excision. A single pedicle flap, sufficiently large to cover completely the raw area, was taken from the lateral nasal fold and the anterior portion of the cheek, partly rotated and advanced into position.

Excess convexities of the dorsum nasi are usually associated with some elongation and drooping of the nasal tip (fig. 4). Corrections are made by freeing the overlying skin and subcutaneous tissues from the cartilaginous bridge, and the periosteum from the bone to be removed, through an incision made within the superior portion of the anterior naris. In most instances I prefer to remove the excess convexity by means of numerous rasps, in assorted shapes and sizes. Bone dust is removed with meticulous care, no rasp having been used is reinserted into the field of operation until it has been carefully brushed and cleansed in sterile water. In some instances saws are preferred in removing very large humps. In most cases the width of the new dorsum can be suffi-

ciently narrowed by rasping the anterolateral portions of the frontonasal processes of the maxillas while they, and the remnants of the nasal bones, are being rounded and smoothed. If the width of the new dorsum is too great, the frontonasal processes may be divided with a saw, or narrow

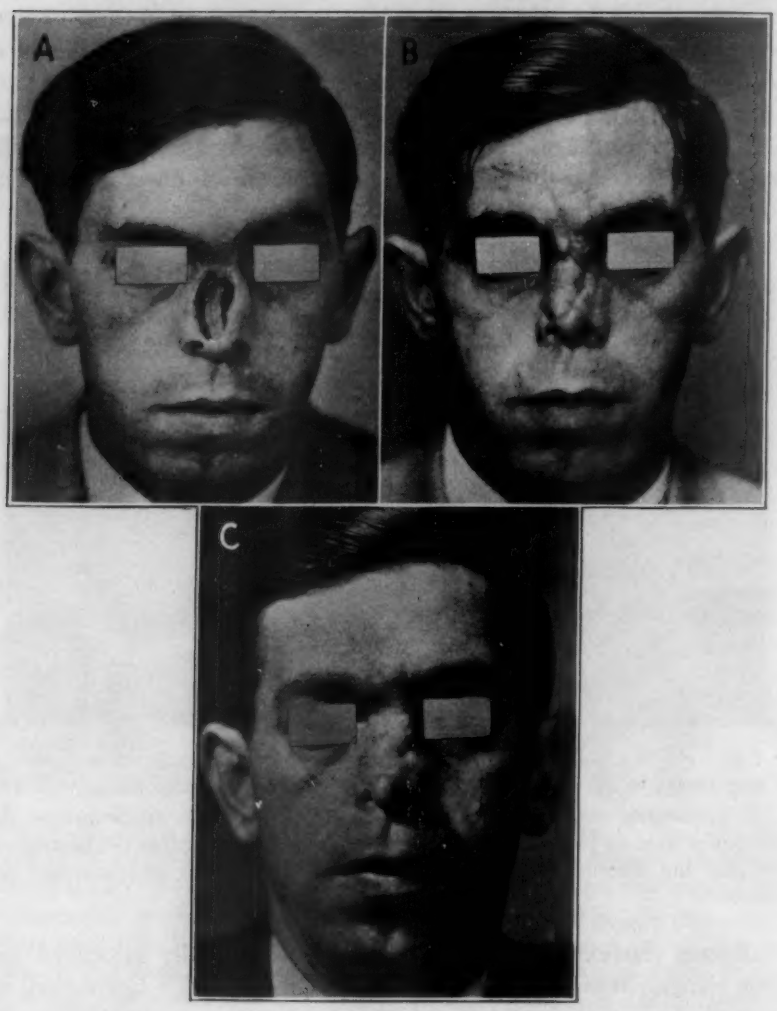

Fig. 8 (R. H., aged 27).—*A*, destruction of the dorsum of the nose by an arsenic paste, used by an advertising cancer quack to remove a wart.

B, appearance seven months after primary repair by use of a delayed double-epithelized pedicled frontal flap.

C, appearance seven years after secondary operation to minimize scars at the margins of the graft.

chisel, near their attachment to the bodies of the maxillas and then pressed medially to the desired width. After the columella is freed from

its attachment to the septal cartilage by a through and through incision, a triangular section of the cartilage of the necessary size to secure the desired degree of shortening is removed and the columella then sutured to the septum in the position which properly elevates the tip.

A rhinophyma, even of marked degree (fig. 5), may be trimmed down to approximately normal size and contour by multiple deep elliptic excisions, the nodules being first removed and then as much of the remaining redundant tissue as the individual case may require. When the margins of the skin are approximated, healing is surprisingly rapid and the scarring relatively inconspicuous. Two operations, one or two months apart, are usually required. Should some undue thickening of the epidermis still remain around some of the larger pores, it can be minimized

Fig. 9 (N. F., aged 53).—Appearance twenty-two months after excision of nearly the entire nose because of destruction by a basal cell epithelioma, which had not responded to irradiation. The bases of the alae, the lower portion of the columella and the extreme superior portion of the bony structures were allowed to remain. The area was repaired by use of a delayed double-epithelized pedicled frontal flap, cut to fit a previously prepared pattern. (Unfortunately, through a misunderstanding between the tumor clinic and the maxillofacial department, no preoperative photograph was made.)

by a series of applications of a saturated solution of trichloroacetic acid over several small areas at one time, and repeated over other similar areas at ten day or two week intervals until the surface is fairly smooth and even.

Loss of external nasal soft tissue is preferably repaired by skin from nearby areas, because the color and texture match more closely than if the skin is taken from any distant point. If the area is small, the sur-

rounding tissue may be undermined and advanced. If slightly larger (fig. 6), then immediate or delayed pedicled flaps from the nasolabial folds, which are very mobile and have splendid blood supply, are used (fig. 7). To repair large deficiencies, delayed frontal flaps, either single- or double-epithelized, as may be required, are most satisfactory (figs. 8 and 9).

SUMMARY

Ill advised, inaccurate articles on plastic surgery in lay publications are deplored because of the tendency which they exert toward increasing self consciousness in many hypersensitive persons with insignificant deformities. Operations on such persons seldom increase their happiness or decrease the introspective tendencies.

Several types of deformities have been presented. In planning plastic reconstruction, one should visualize the ultimate result desired and outline accordingly the various steps necessary in the repair. Knowing the number of contemplated operations and the probable intervals helps the patient in arranging adequate time for operations and convalescence.

Accuracy in outlining flaps, gentleness in handling tissues and evenness in the approximation of subcutaneous tissues and skin margins, all tend to minimize scar tissue and thereby assist in securing the best functional and cosmetic results.

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GUNSHOT LESIONS OF THE LARYNX

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There is little to be found in the literature on gunshot wounds of the larynx. The data available deal as a rule with reports of a stab or cut in the neck, the clinical picture of which differs largely from that of gunshot wounds of the larynx. Perichondritis and chondritis of the laryngeal cartilages caused by a gunshot wound display certain peculiarities and demand therapeutic measures different from those for perichondritis of other origin. It may be mentioned in passing that in the great majority of cases wounds of the upper respiratory passages and of the organ of hearing are combined with lesions of adjacent regions.

My observations in various cases, in which the injury lasted five days to six months, offer a fairly complete clinical picture from the initial injury to the concluding stage. It is not intended to present here a classification of typical forms of laryngeal lesions which I have observed during the present war. This phase of the subject will be discussed elsewhere. I shall consider here only the lesions that have been of most frequent occurrence in my experience. These may be listed as follows:

Injury limited to the skin of the frontal surface of the neck caused by a tangential bullet or shell fragment.

Open or subcutaneous wound of the thyroid cartilage. Such an injury is soon followed by recovery. The wound closes promptly, and the respiratory and phonatory functions are preserved.

Gunshot wound involving injury of the thyroid cartilage, which results in the development of chondritis and perichondritis.

Shattering of part of the cricoid cartilage.

Impaired integrity of all laryngeal cartilages.

Fracture of one or several tracheal rings, the thyroid cartilage and the hyoid bone.

Injury of the larynx and damage of cervical vertebrae.

The peculiarities of the clinical picture of traumatic perichondritis of the laryngeal cartilages call for consideration. I should mention first of all the sluggish course of perichondritis of these cartilages due to a gunshot wound. The general phenomena as a rule are relatively slight unless the perichondritis is complicated by sepsis or inflammation of the tracheobronchial system. Changes in the soft tissues of the frontal surface of the neck are frequently extremely slight; often there is only slight tenderness to touch. Morphologic changes in the blood are insignificant, and the blood sedimentation test may be normal.

The laryngoscopic picture varies according to the location of the process and the degree of the injury. Sometimes incomplete opening of the mouth prevents examination of the larynx. In such cases tracheotomy is of greatest usefulness; sometimes this method is the only possible one. In this way I have sometimes succeeded in determining the character and localization of tissue changes in the larynx.

Lesions of the trachea and larynx often require tracheotomy. Prolonged wearing of the metallic tracheotomy cannula may be necessary, and this may influence the course of the injury. As a matter of fact, irritation of the surface of the wound caused by the tracheotomy tube may either aggravate or prolong the inflammatory process. It is a well known fact that the presence of a tracheotomy tube favors the initiation of an inflammatory process in the lungs. Under hospital conditions, in which constant otolaryngologic assistance is available, the tracheotomy cannula is removed a few days after operation, provided only the trachea, and not the larynx, is injured.

A grave complication of gunshot injury of the larynx and trachea is pneumonia, resulting either from the lesion itself or from surgical intervention. A lethal issue is not rare. During recent months no case of inflammation of the lung has been recorded, notwithstanding numerous operations which might have been expected to cause pneumonic complications. During the first days following operation one of the sulfonamide compounds is administered, every six hours on the first day and every eight hours on the second and third days.

The majority of authors advise that in cases of perichondritis and chondritis of the larynx surgical intervention should always be postponed until all acute inflammatory processes have subsided, usually six months. A different view has been expressed by some authors (Zitovich). To quote Professor Ivanov, an authority on laryngeal stenosis:

> In cases in which laryngostomy was performed more than a half-year after the laryngeal lesion and the temperature had been normal for a long period and painful sensations were absent, I have often found suppurative foci in and around the larynx; pus had accumulated between the cartilage and the intact perichondrium, separating them and thereby leading to extensive lesions. Had the suppurative foci been opened much earlier, structural changes would probably have been less extensive.

I have seen similar cases. In the majority of my cases chronic stenosis of the larynx has been prevented by early surgical intervention.

I wonder what are the theoretic grounds of those surgeons who recommend expectant treatment of laryngeal perichondritis. In cases of this condition surgical treatment usually is carried out very late, when the skeleton of the entire larynx, or a considerable part of it, is destroyed. The lumen of the larynx is closed completely, and the patient has to

wear the tracheotomy tube permanently, or he is subjected to repeated surgical operations for the purpose of decannulation.

Surgical intervention, therefore, is to be recommended in the early stages of a gunshot lesion of the larynx, with a view not only to preserving as much as possible of the skeleton of the larynx, but to avoiding sepsis due to fragments of infected tissue adhering to the soft tissues.

It is surprising to see how little attention is usually paid to roentgenography, which is a valuable method in the diagnosis of gunshot lesions of the larynx. In my opinion this means is indispensable.

The clinical picture of a gunshot lesion of the larynx is sometimes obscured by a simultaneously occurring fracture of the hyoid bone. During a comparatively short time I have recorded 40 cases in which a lesion of the larynx was associated with such a fracture. The symptoms were pain, expectoration of blood (due to injury of the pharyngeal mucosa), disturbance of phonation, pain on extending the tongue or on shifting it in a direction contralateral to the fracture, and immobility of the epiglottis, together with the roentgenographic evidence of the lesions.

My experience with gunshot lesions of the larynx leads to the following conclusions:

1. Perichondritis of the laryngeal cartilages caused by a gunshot wound often runs a sluggish course.

2. In some cases, after cicatrization of both the wound of entrance and the wound of exit, suppuration develops in the deeper tissues.

3. With hospitalized patients who have been tracheotomized decannulation should be performed as soon as possible.

4. Early external operation is advisable when the laryngeal framework has been injured, the kind of operation being varied according to conditions present.

5. With surgical intervention the best control of infection is afforded by the use of one of the sulfonamide compounds.

6. In a patient with a gunshot lesion of the larynx the condition of the hyoid bone should always be determined.

7. Roentgenographic examination of the larynx is a valuable aid in the diagnosis of gunshot wounds.

RATIONALE OF ESTROGEN THERAPY OF PRIMARY ATROPHIC RHINITIS (OZENA)

RELATIONSHIP OF THE PHARYNGEAL PITUITARY TO OZENA

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The establishment of the differential diagnosis of ozena as primary atrophic rhinitis of vascular origin and secondary atrophic rhinitis of infective source and the presentation of a definite pathologic picture for each condition have made possible a scientific approach to the therapy.

At the Third International Rhinolaryngological Congress, held in Madrid in 1932, I had the privilege of demonstrating a series of pathologic photomicrographs on the basis of which the heterogeneous group of conditions characterized by shrinkage of the expansile tissues of the nose, with increase in the nasal space, associated with fetid crusts, and commonly called synonymously atrophic rhinitis and ozena, could be separated pathologically into two subtypes. One of these (atrophic rhinitis, or secondary atrophic rhinitis) presented the pathologic picture of inflammatory reaction to infection that had invaded the tunica propria of the mucosa; the other, ozena, or primary atrophic rhinitis, was due to changes in the blood vessels.

In the second type I demonstrated a pathologic picture of vascular disturbance relatively free from infection, with pronounced thickening of the media of the arteries, leading in some instances to true endarteritis. There is an associated loss of the expansile mechanism of the nasal mucosa with erosion of the superficial mucosa and formation of crusts. In the secondary atrophic rhinitis one sees no involvement of the blood vessels, but rather round cell infiltration of the tunica propria, with chronic inflammatory processes disturbing the distensile mechanism of the nasal mucosa.

Fortunately, the pathologic picture is further supported by a sharp roentgenographic differentiation, which I described. In all the cases of secondary atrophic rhinitis rarefaction of the bone was present throughout, with a thin vomer and a fragile lateral nasal wall, whereas in cases of the primary atrophic type the bony structure of the septum, the lateral nasal wall and the sinus walls were definitely sclerotic. This made possible an easy differential diagnosis without resorting to tissue sections.

As an example of the confusion that existed prior to my pathologic differentiation of secondary and primary atrophic rhinitis (ozena), I

quote a paragraph from Lautenschläger's article in Denker and Kahler's "Handbuch."

A further error existed in the assumption that in atrophic rhinitis the entire bony framework was atrophic. Operation on the sinuses reveals quite the contrary

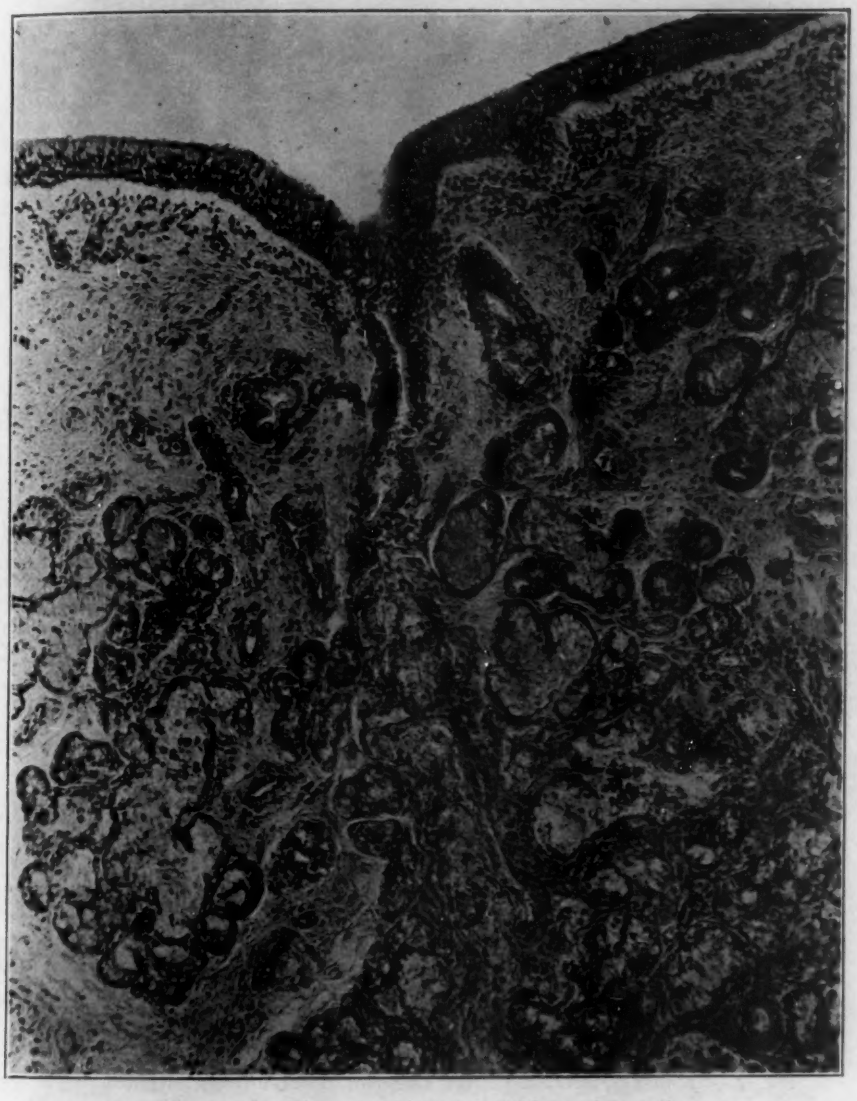


Fig. 1.—Anterior tip of inferior turbinate (normal).

in most cases of advanced ozena, namely, great thickening and sclerosis of the bony walls of the sinuses, particularly the medial wall of the maxilla. Undoubtedly, these bony changes follow inflammation of the mucosa. A thickened, hyperplastic, congested sinus lining is associated usually with a correspondingly thin bony wall,

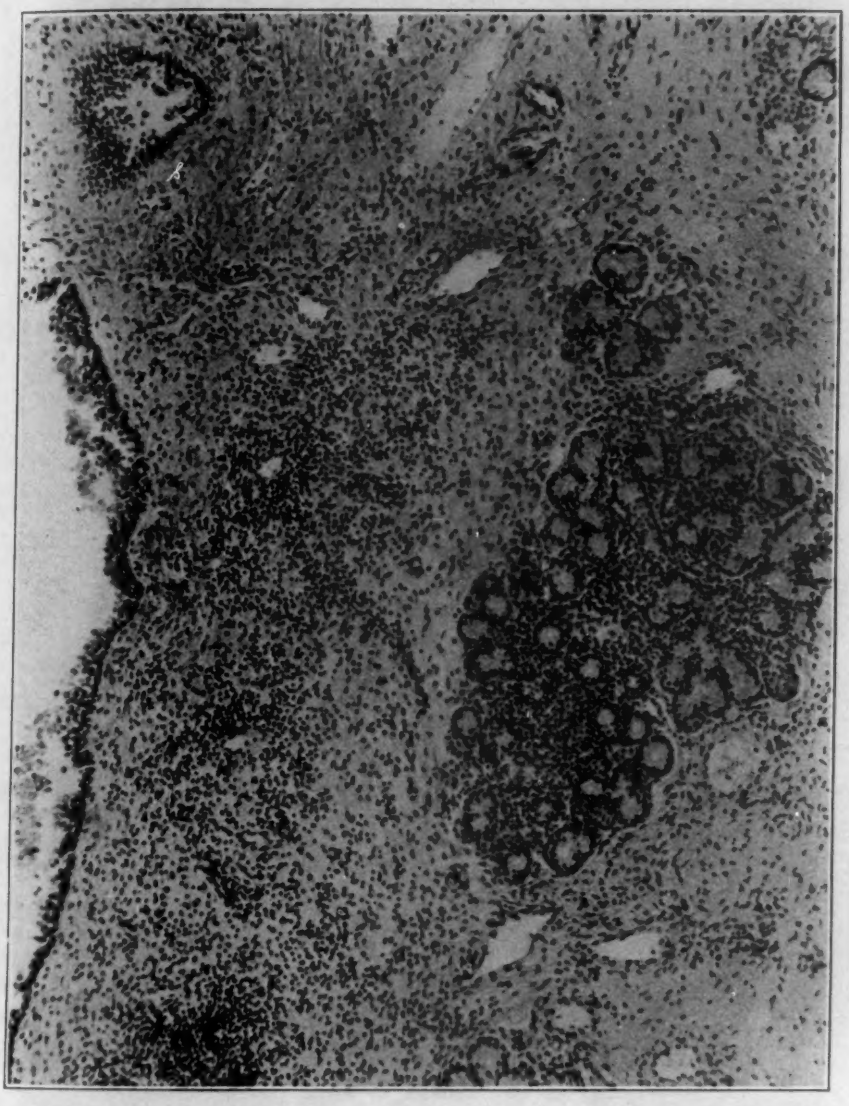
while an atrophic, fibrous sinus mucosa as a rule is associated with a strong, thickened bony wall. It is possible that in advanced age the thickened lateral bony wall may also gradually become atrophic, since one finds in old people with ozena little thickening of the wall. The case described by Bergeat was that of a young person

Fig. 2.—Secondary atrophic rhinitis (anterior tip of inferior turbinate).

in whom the whole bony structure of the nose, including the lateral wall, consisted only of thin bony lamellas.

In Lautenschläger's description one sees no differentiation between secondary and primary atrophic rhinitis (ozena) as separate diseases.

He, therefore, found it difficult to correlate the thin bony structure in Bergeat's case, which was apparently an instance of secondary atrophic rhinitis of infectious origin, with the sclerosed bony walls in the cases he



Feb. 3.—Secondary atrophic rhinitis (anterior tip of middle turbinate).

described, which were instances of true primary atrophic rhinitis. He also failed to recognize that the changes in the blood vessels were specific. Thus, he stated in the chapter on histologic changes:

> Concerning the changes in the blood vessels there are many contradictory reports. Some authors have stated that the vessels were practically unchanged, or they found

them widened and unthickened; the majority saw only the connective tissue thickening of the adventitia, while still others observed thickening of the adventitia and media. E. Frankel, Schönemann and H. Krause mentioned also the endarteritis.

Fig. 4.—Primary atrophic rhinitis, or ozena (middle turbinate).

The fact that secondary atrophic rhinitis is associated with unchanged or dilated vessels and ozena (primary atrophic rhinitis) is characterized by thickening of the media of the arteries, with at times obliterating endarteritis, was not observed. The clarification of the entire picture followed the differential diagnosis here described. Separation of the vascular primary atrophic rhinitis (ozena) as a disease separate and

apart from the infectious secondary atrophic rhinitis should be made clinically, as well as pathologically and roentgenographically.

Interestingly, the illustration (fig. 10) showing the posterior rhinoscopic view in a case of ozena in Lautenschläger's article is a striking

Fig. 5.—Primary atrophic rhinitis, or ozena (anterior tip of middle turbinate).

example of vascular tissue changes and should have led to a better understanding of the pathologic picture. It is reproduced in this paper because it demonstrates the changes in the mucosa of the turbinate, with pallor on one side and engorgement on the other. The alternating character of this pallor is a pointed argument for its vascular origin. The illustration

also shows the characteristic crust formation and pallor of the epipharynx, particularly at the junction of the septum and the sphenoid bone. I have found this area uniformly affected in cases of ozena, and I propose to correlate involvement of this area with the pharyngeal pituitary and to establish the hormonal relation of this area to ozena.

Fig. 6.—Primary atrophic rhinitis, or ozena (middle turbinate).

In a memorandum to the Imperial Academy of Vienna in 1904, Erdheim first noted the existence in the mucosa of the pharyngeal roof, in the newborn as well as in the fetus, of hypophysial tissue, to which he gave the name pharyngeal pituitary. Three years later Arai made the same observation in animals as well as in man. Since then

the pharyngeal pituitary has been mentioned regularly in textbooks on human anatomy. Much interesting work has been done on this subject by Civalleri (1907), Haberfeld (1909), Poppi (1911) and Citelli (1911, 1912, 1913).

Fig. 7.—Primary atrophic rhinitis, or ozena (middle turbinate).

ANATOMIC RELATIONS

The pharyngeal pituitary seems to be a constant formation. Haberfeld found it consistently in 51 cadavers (8 fetuses, 11 newborn infants, 7 infants and 25 adults) which he examined for this purpose, although the dimensions and positions varied. More recently Melchionna found it in 48 of 52 adult cadavers he examined.

The pharyngeal pituitary occurs in the depths of the mucosa which covers the inferior surface of the basisphenoid and forms the roof of the pharynx, lying in the midline. It is most often observed on that part of the body of the sphenoid bone which lies just behind the vomero-sphenoid articulation, at the level of the inferior orifice of the cranio-pharyngeal canal when that canal is present. Rarely it is seen on the posterior border of the choanal septum—sometimes on the superior, sometimes on the middle and sometimes on the inferior portion. Haberfeld in 1 case found it lower still, on the anterior border of the soft palate.

Fig. 8.—Roentgenogram in a case of primary atrophic rhinitis (ozena).

The pharyngeal pituitary is ordinarily seen as a sagitally elongated body, sometimes cylindric, sometimes flattened from side to side and sometimes flattened superoinferiorly. It is directed from before backward and upward. Its dimensions, especially its length, are extremely variable, the length ranging from 2 to 5 mm., the width from 1 to 3 mm. and the thickness from 0.25 to 0.50 mm.

The pharyngeal pituitary lies in the fibrous connective tissue which separates the pharyngeal mucosa from the base of the skull. Its anterosuperior, or cranial, surface is in relation to the base of the sphenoid, being separated from it by fibrous tissue and periosteum. Its posteroinferior, or pharyngeal, surface looks down into the pharyngeal cavity, being separated from it by mucous glands and epithelium. The

superior, or proximal, end is in relation to the base of the skull, which it may or may not reach. Sometimes this upper end may even lie embedded in the inferior portion of the craniopharyngeal canal. The inferior, or distal, end is ordinarily encountered in the neighborhood of the most posterior part of the vomerosphenoid articulation. In cases in which the body is greatly elongated this end may lie in contact with the posterior margin of the choanal septum. The area immediately surrounding the structure, especially its anterosuperior surface, is rich in small vessels and nerves. These vessels appear in two groups, one which runs toward the vomerosphenoid articulation and another which

Fig. 9.—Roentgenogram in a case of secondary atrophic rhinitis.

runs upward to the site of the craniopharyngeal canal, and into it when it exists. The pharyngeal pituitary is always situated in front of Luschka's pouch and in front of the adenoid. Its relation to the latter varies with the age of the person. In the newborn the adenoid is small and lies behind it. In the adolescent and in the adult the adenoid enlarges, and it may then completely envelop the pharyngeal pituitary. In old age the adenoid atrophies, and then the condition found in the newborn again prevails.

EMBRYONIC DEVELOPMENT

The embryonic development of the pharyngeal pituitary may be summarized as follows: The buccal fossa, or stomodeum, sends a long

diverticulum upward and backward, known as Rathke's pouch. The superior portion becomes spherical and forms the hypophysial cecum. The inferior portion remains narrow, and, lying within the craniopharyngeal canal, becomes the hypophysial pedicle. The pedicle runs from the intracranial cecum to the pharyngeal mucosa. The cecum forms the glandular organ, the anterior lobe of the pituitary, while the pedicle disappears. The incomplete disappearance of the pedicle gives rise to three possibilities: (1) an accessory hypophysis in the floor of the sella turcica, an intracranial pituitary; (2) an accessory hypophysis in the craniopharyngeal canal, an interosseus pituitary, and (3) an accessory hypophysis in the pharyngeal mucosa, a pharyngeal pituitary. This pharyngeal pituitary develops as glandular tissue, just as the anterior lobe does, and it may even supplant the anterior lobe.

MORPHOLOGIC CHARACTER

The morphologic structure of the pharyngeal pituitary is exactly the same as that of the anterior lobe, with chromophobe and chromophil cells and colloid in follicles.

The pharyngeal pituitary is surrounded by a neurovascular net, which is extremely well developed, especially on its anterosuperior surface. The arteries penetrate the depth of the gland, following the septum formed by the inward prolongations of the surrounding capsule. The caliber of these vessels depends not on age, as Haberfeld stated, but, as Citelli indicated, on the cause of death and on the pathologic changes which have taken place in the rhinopharyngeal region.

The circulatory relationship between the vascular supply of the pituitary and that of the nasopharynx has already been described by Citelli. By injecting india ink into the torcular Herophili of 20 cadavers and studying the heads both microscopically and macroscopically, he was able to show a direct anastomosis between the vessels of the floor of the sella turcica and those of the pharyngeal roof.

PRESENT INVESTIGATION

The intimate relation of the vascular supply of the nasal and that of the nasopharyngeal mucosa suggested also a direct hormonal action of the pharyngeal pituitary on the nasal mucosa. With this in mind, I undertook the study of the physiologic properties of the pharyngeal pituitary. For this purpose the nasopharynx of the ox was found most suitable because on section it showed a favorably sized pharnygeal pituitary. Twenty-five freshly killed ox heads were quickly stripped of the nasopharyngeal mucosa covering the anterior half of the nasopharynx.

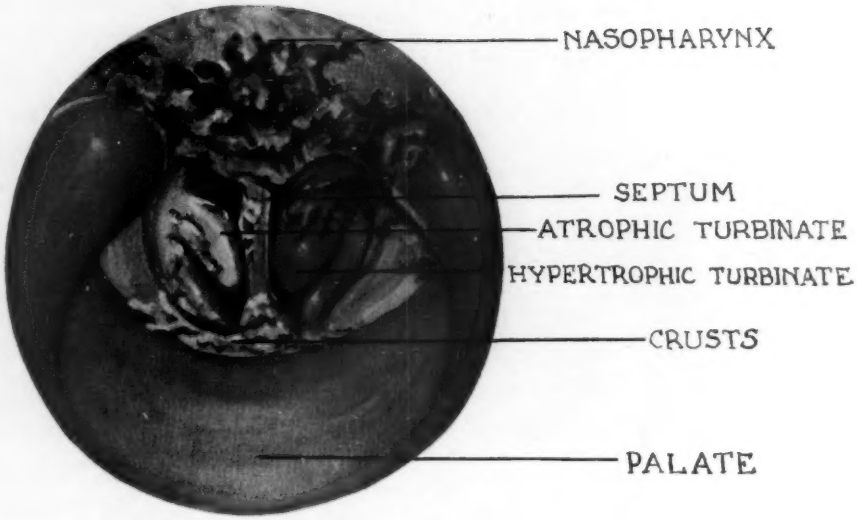
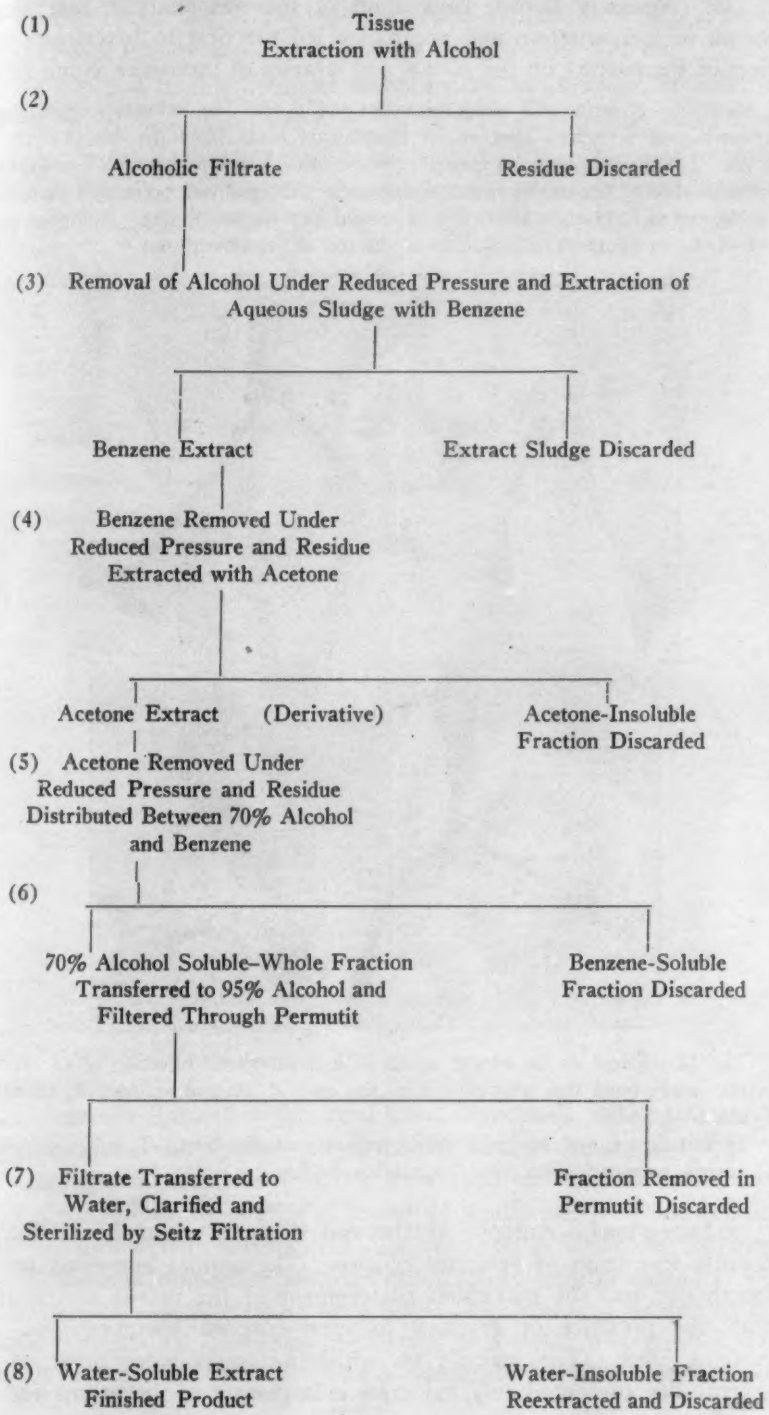


Fig. 10.—Ozena (from Lautenschläger, A.: Die Rhinitis atrophicans, in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohren-Heilkunde, Berlin, Julius Springer, 1926, p. 604).

On removal they were immediately placed in a thermos jar and removed to the laboratory, where extraction was carried out as follows:


(1) Tissue
Extraction with Alcohol
(2)
Alcoholic Filtrate
Residue Discarded
(3) Removal of Alcohol Under Reduced Pressure and Extraction of Aqueous Sludge with Benzene
Benzene Extract
Extract Sludge Discarded
(4) Benzene Removed Under Reduced Pressure and Residue Extracted with Acetone
Acetone Extract (Derivative)
Acetone-Insoluble Fraction Discarded
(5) Acetone Removed Under Reduced Pressure and Residue Distributed Between 70% Alcohol and Benzene
(6)
70% Alcohol Soluble–Whole Fraction Transferred to 95% Alcohol and Filtered Through Permutit
Benzene-Soluble Fraction Discarded
(7) Filtrate Transferred to Water, Clarified and Sterilized by Seitz Filtration
Fraction Removed in Permutit Discarded
(8) Water-Soluble Extract Finished Product
Water-Insoluble Fraction Reextracted and Discarded

The commonly known congestion of the nasopharynx and nasal mucosa in menstruation and pregnancy led me first to investigate the effect of the extract on the uterus and ovaries of immature white mice.

Material.—A series of 8 white mice was employed. The animals received daily intraperitoneal injections of 1 cc. of the extract made from the nasopharynx of the ox. The animals were all immature white mice, weighing between 7 and 8 Gm. Animal 1 died at the end of twenty-four hours. Autopsy was performed on animal 2 at the end of forty-eight hours and on animal 3 on the fourth day. Autopsies were carried out on the remaining animals at the end of the seventh day.

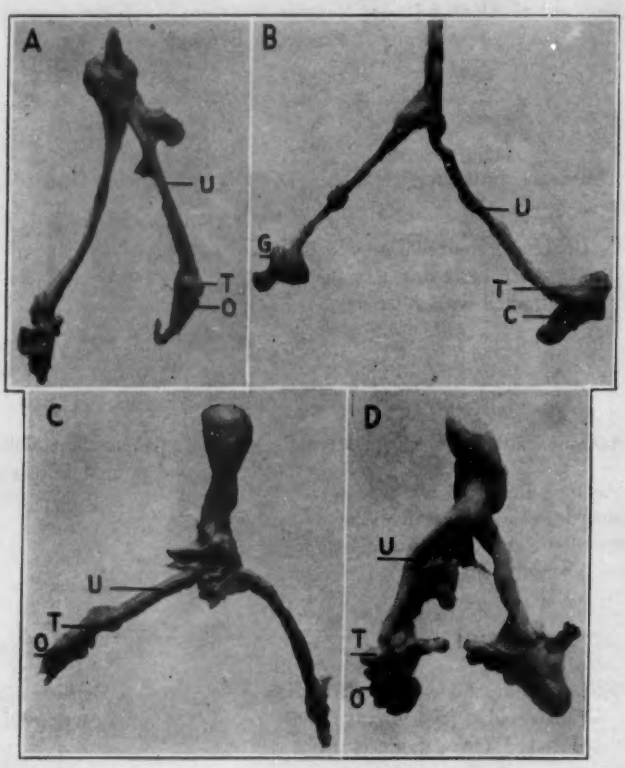


Fig. 11.—Effect on the mouse uterus of intraperitoneal injection of an estrogen extract made from the nasopharynx of the ox. *A*, control animal; *B*, animal 1; *C*, animal 2, and *D*, animal 4.

In this figure, and in figure 12, *U* indicates uterine horn; *T*, tube; *O*, ovary; *C*, corpus haemorrhagica, and *G*, graafian follicle.

Observations.—Autopsy at the end of twenty-four hours showed definite formation of graafian follicles. The animal autopsied on the fourth day had the maximum enlargement of the uterus and ovaries, with the presence of graafian follicles, corpora haemorrhagica and corpora lutea. The animals on which autopsies were done on the seventh day presented only moderate enlargement of the uterus and the

presence of follicles. The maximum influence was apparently attained at the end of the fourth day, and the initial indications of action on the gonads was already visible at the end of twenty-four hours.

In addition to this influence demonstrable at autopsy, the reactions of the animals following the injection of the tissue extract were interesting. In about ten minutes after injection there were signs of increased activity; the mice receiving injections were more lively than the controls and showed an increased interest in food. There was also evidence

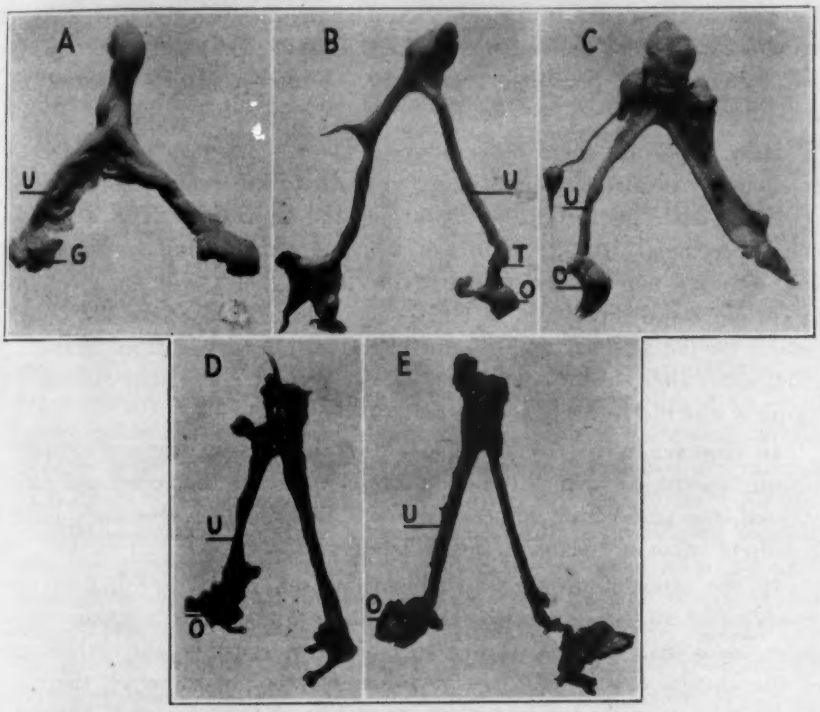


Fig. 12.—Effect on the mouse uterus of intraperitoneal injections of an estrogen extract made from the nasopharynx of the ox. *A*, animal 4; *B*, animal 5; *C*, animal 6; *D*, animal 7, and *E*, animal 8.

of an increase in growth of the facial hairs, particularly in the animals on which autopsies were performed at the end of the seventh day.

Although this series is relatively small, and such factors as the nonspecific reaction of the ovaries of immature white mice to tissue extracts must be considered, nevertheless, the response is deserving of some consideration in connection with the other phases here presented.

It thus became apparent that a hormone mechanism related to the pituitary gland and possessing estrogenic properties is a normal constituent of the nasal mucosa. The observation also allows a degree of

speculation that, since the pituitary gland is concerned with blood vessel tone, the pronounced thickening of the blood vessel walls shown in the pathologic picture of primary atrophic rhinitis is the end result of tonic overaction of the vasoconstrictor fibers, caused perhaps by a deficiency of the pharyngeal pituitary. The occurrence of nasal and nasopharyngeal congestion during pregnancy also suggests the possibility that the pharyngeal pituitary exercises an active vasodilator influence on the nasal mucosa. This is all the more likely since the demonstration of the usefulness of estrogen in cases of ozena by Mortimer. The relationship of the nasopharynx to primary atrophic rhinitis may also be seen in the constant occurrence of crusting, pallor and, in some instances, scarring in the region of the pharyngeal pituitary. Lautenschläger's illustration also shows this.

How important these observations are for therapy is now evident. If all forms of atrophic rhinitis are grouped together and called ozena confusion and therapeutic failure are bound to result. The atrophic rhinitis secondary to old rhinitis or sinusitis complicating scarlet fever will certainly not respond to estrogenic therapy, nor will atrophy due to chronic nasal diphtheria in childhood be benefited. Here the blood vessels are not affected, and in some of the sections new-formed granulations are visible. The denudation of the ciliated epithelium shown in figure 3 was the result of a deep-seated slough.

If, however, estrogenic therapy is used in cases of primary atrophic rhinitis (ozena), in which the blood vessel walls are thickened and constricted, the vasodilator influence incident to estrogenic stimulation has a definite value and provides desirable effects.

In the cases of primary atrophic rhinitis a number of degrees of involvement must be expected. It would be a mistake to consider only those cases in which the classic trio of fetor, crusting and shrinkage of the mucosa exists, for cases of milder form occur in which there is no fetor or crusting but loss of expansibility of the mucosa is present. The milder form is much more frequent than the severe and occurs chiefly in patients in the menopause. The chief complaint in cases of this type is dryness of the nasopharynx and nose, with at times a choking feeling in the throat, frequently diagnosed as globus hystericus. The patients apparently benefit much in relief from these symptoms from nasal and parenteral estrogen therapy.

Three types of medication were tried: (1) a nasal spray of estrogen in oil alone; (2) a nasal spray of estrogen in oil combined with parenteral injections, and (3) oral administration of estrogen tablets. My observation was that the combined nasal spray and parenteral injection of estrogen in oil was the most effective. For the nasal spray I used

amniotin in oil (estrone in oil) and injected 2,000 international units biweekly. In another group of cases α-estradiol dipropionate was used, 0.2 mg. biweekly. The two preparations seemed equally effective. Many case reports are now already in the literature, and repetition is unnecessary. It is worth while, however, to report on the use of diethylstilbestrol in oil, 10,000 international units of estrogen per cubic centimeter, in 1 case (Mrs. C.), in which it was employed for three months. During this time the patient used intranasally as drops three 10 cc. vials, a total of 300,000 international units. There were no unpleasant by-effects, no nausea, no dizziness and no circulatory disturbances. The nasal mucosa appeared to be definitely improved, and the patient has not required more than occasional observation in over a year. The crusting had diminished, and there is little nasal discomfort. One may thus find in diethylstilbestrol a further aid in the therapy of primary atrophic rhinitis (ozena).

It thus becomes clear that the first essential in therapy is the differential diagnosis of primary atrophic rhinitis (ozena) of vascular origin and secondary atrophic rhinitis resulting from a severe nasal infection. This can be accomplished by examination of the tissue specimen, which shows in the case of primary atrophic rhinitis endarteritis and fibrosis and in the case of secondary atrophic rhinitis round cell infiltration and no involvement of blood vessels. Or it can be accomplished by roentgenographic examination, which shows sclerosis and thickening of bone in cases of primary atrophic rhinitis (ozena) and rarefaction and thinning of bone in cases of secondary atrophic rhinitis. The primary atrophic rhinitis is apparently related to involvement of the pharyngeal pituitary and the pituitary gland and is amenable to treatment with estrogenic substances. Extracts of the nasopharynx also possess estrogenic properties. Treatment of secondary atrophic rhinitis should be studied from the point of view of determining the infective agent and using specific remedies when possible. Use of the sulfonamide compounds appears indicated in some cases of this type. Estrogens would obviously be ineffective.

CONCLUSIONS

The differential diagnosis of primary atrophic rhinitis (ozena) and secondary atrophic rhinitis, as previously described, is essential in the proper use of estrogenic therapy.

The role of the pharyngeal pituitary and its relationship to primary atrophic rhinitis (ozena) is considered.

A bibliography on the pharyngeal pituitary is added for ready reference.

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MANIFESTATIONS OF DERMATOLOGIC DISEASES IN THE UPPER RESPIRATORY TRACT

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ST. LOUIS

The chief purpose of this contribution is to stimulate more interest among laryngologists in manifestations of dermatologic diseases in the respiratory tract. This group of pathologic processes have never been allotted the space they deserve in the otolaryngologic literature, although they are constantly appearing in cases under the observation of otolaryngologists. Their description and treatment are much more adequately covered in the dermatologic literature. In fact, the most extensive presentation of this subject I could find published in the otolaryngologic literature during the past ten years was by a dermatologist, Dr. Hamilton Montgomery.

I shall list as many of the diseases having exhibitions pertaining to the subject as feasible and attempt to describe some of the more important ones. The following dermatologic diseases are reported to exhibit mucosal lesions of the respiratory tract that should be of interest to the otolaryngologist: lichen planus, lupus erythematosus, psoriasis, acanthosis nigricans, keratosis follicularis (Darier's disease), erythema multiforme, dermatitis herpetiformis (Duhring's disease), pemphigus, epidermolysis bullosa, scleroderma and porokeratosis (Mibelli). Owing to the fact that a positive diagnosis of the mucosal lesions in these conditions may hinge on an accurate recognition of the true character of the accompanying cutaneous eruptions, some space must be devoted to the description of the latter.

It must not be assumed that the mucosal and cutaneous manifestations of a disease need necessarily be identical in appearance and character. They may be entirely dissimilar. The constant maceration of a lesion on a mucous membrane from the ever present moisture due to secretions often containing a high percentage of digestive ferments will alter its appearance from what it would be on dry skin. In other instances structures are involved on the skin which are not present on mucous membranes and vice versa.

As an illustration of this fact I quote a statement I made pertaining to Darier's disease in an early draft of this paper and retracted on

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the final revision. I failed to take into consideration that the same disease can break out in different tissues with entirely different lesions. The statement was as follows: "Keratosis follicularis being what it is (i.e., a disease of the epidermis occurring largely but not entirely about follicles, hair, sebaceous or sweat, with acanthosis, benign overgrowth of the prickle cell layer of the epidermis and thickening of the stratum granulosum) makes it hard for one of my elemental knowledge of the subject to visualize just what is involved when the mucous membranes are attacked."

Furthermore I cannot conscientiously defend against criticism the use of the words "dermatologic disease" in questionable instances, because very often a so-called dermatologic disease or a mucosal disease is merely a manifestation of a systemic disease. Either may be just another clinical symptom, like fever.

LICHEN PLANUS

The mucous membranes are commonly invaded by lichen planus, and not infrequently the mucosal lesions precede the appearance of the cutaneous eruption. In fact, typical cases of mucosal involvement without any accompanying cutaneous manifestations have been reported. Ormsby[1] stated: "Dubreuilh believes that more cases of involvement of mucous membranes occur without cutaneous lesions than of the last named without mucous symptoms."

The characteristic cutaneous lesions of lichen planus are multiple angular-edged papules of various shades of crimson, purple or violaceous color, becoming darker as they grow older. The elevations are usually flat topped but may have umbilicated, rounded, bluntly pointed or even acuminate summits. Coalescence of lesions may form elevated ridges, plateaus or plaques with roughly perpendicular sides. The tops of the flat papules often present an opaque, bluish, silvery sheen, which on minute examination may show small lines, known as Wickham's striae. Typical papules are usually 1 to 10 mm. in diameter and are often arranged in linear fashion as though following a nerve or scratch mark. The cutaneous lesions are predominantly on the flexor surfaces of the extremities, and the wrists seldom escape. The disease is chronic, and recurrences are common even with treatment.

The bullous lesions, annular forms and other rare manifestations of the disease cannot be described here.

The most typical mucosal lesions of lichen planus are probably found on the buccal mucous membranes and consist of a retiform arrangement of silvery white infiltrated lines or elevated ridges, anas-

1. Ormsby, O. S.: A Practical Treatise on Diseases of the Skin, Philadelphia, Lea & Febiger, 1915.

tomosing with one another to form lacelike figures, interspersed here and there with small infiltrated spots, arcs, rings or raised dots. Delicate threadlike or spinelike projections may be seen extending outward from any type of lesion and often result in transforming some of the rounded mounds into irregular stellate configurations. The infiltrated lesions are distinctly palpable, and the linear ridges may roll under the palpating finger, like a small elastic rubber band embedded in the mucosa. Mucosal lesions of lichen planus are not infrequently mistaken for a potentially dangerous leukoplakia. Horrible mutilations from actual thermocautery have been done in cases of this harmless condition.

LUPUS ERYTHEMATOSUS

The mucosae and mucocutaneous junctures are occasionally involved in lupus erythematosus, which is a systemic disease (usually chronic and nondangerous to life but occasionally acute and fatal) characterized by erythematous macules or scaly plaques covering discoid areas on the malar regions of the cheeks and connected over the nose by a narrower strip which sometimes sends a flaplike extension downward to the nasal tip. Occasionally it is atypical, i.e., limited to areas not usually affected, or asymmetric, only one cheek being involved. The disease is predominantly on exposed surfaces, and outbreaks have often been recorded following severe sunburn.

Some cases of acute fatal "butterfly fever" or "butterfly disease" have made the front pages of the St. Louis newspapers recently.

The mucous membranes of the lips and mouth are usually selected, the predominant lesions being of the discoid type and differing in appearance from the cutaneous lesions only because of the difference in character of the involved tissues. The buccal mucosa is often affected, the lesions appearing as slightly elevated, well circumscribed plaques with sharply marginated bluish red borders and centers usually covered with a white thickened epithelial layer but sometimes raw, eroded or superficially ulcerated. The entire lip may be involved, being swollen and bluish red at first but later becoming covered with whitish adherent scales resembling peeling collodion. On the vermilion border of the lips the same discoid type seen in the buccal mucous membranes may appear, or only a narrow, silvery white strip near the cutaneous margin, resembling a fringe of silver, may be found, which has been referred to as "silvering of the lips."

The diagnosis of mucosal lupus erythematosus may depend on recognition of the accompanying cutaneous eruption. To demonstrate that this is not always easy: I had a case in which a late syphilid certainly appeared to be lupus erythematosus until the eruption promptly disappeared on vgiorous antisyphilitic treatment after a 4 plus Wassermann reaction was obtained.

PSORIASIS

Dermatologists do not seem to agree among themselves as to whether or not true lesions of psoriasis have actually been found on the mucous membranes.[2]

Schütz [3] reported 2 cases and referred to others in the literature, but Hyde, Ormsby and other dermatologists seem to believe that the lesions on the mucous membrane, including the so-called psoriasis linguae, for instance, are actually manifestations of leukoplakia occurring with psoriasis. In the illustration in Stelwagon's "Diseases of the Skin" [4] labeled "psoriasis of the tongue" the disease certainly has the appearance of leukoplakia, and it was so considered by him.

Becker and Ritchie reported a case of psoriasis of the buccal mucosa associated with characteristic lesions elsewhere and showed a picture of the cheek everted to reveal lesions on the left buccal mucous membrane which they apparently believed to be genuine psoriasis.

Psoriasis is a chronic (rarely acute) inflammatory disease with an eruption showing a predilection for the elbows, knees and extensor surfaces of the joints and limbs, but it may become generalized. The typical mature lesions are variously sized papules, plaques or raised infiltrated areas sharply defined from the surrounding normal skin and covered with silvery white, shiny, mother-of-pearl scales, overlapping or arranged in thin, shale-like layers resembling mica. If the scales are scraped off with a knife, for instance, recent lesions remain covered with a bright red surface over which are scattered small bleeding points, often so minute that they can be seen only through a magnifying lens. The older lesions denuded of scales exhibit a duller red. In the rare rupial form of psoriasis, the silvery scales pile up in layers like a tapering stack of pearly buttons.

ACANTHOSIS NIGRICANS

This is a rare disease characterized by dark brownish gray or sooty-colored pigmentary changes of the skin and mucous membrane. The discolorations of the skin often appear as unevenly raised lines or rounded elevations, papules or plaques, but eventually they may become diffuse and involve large areas causing a resemblance to elephant hide. The furrows of the skin can be distinctly felt by the palpating finger. Two types of the disease are described: One is benign and acute; the other, chronic and usually accompanied by internal malignant lesions, which account for its inevitable fatal termination.

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2. Schlosser, K.: Gibt es eine Psoriasis der Schleimhaut? Dermat. Wchnschr., **100**:387 (April 6) 1935.

3. Schütz, J.: Ueber Leukoplakia oris bei Psoriasis und anderen Dermatosen, Arch. f. Dermat. u. Syph. **46**:433, 1899.

4. Stelwagon, H. W.: Treatise on Diseases of the Skin, ed. 3, Philadelphia, W. B. Saunders and Company, 1904.

The mucous membranes or the mucocutaneous junctions are said to be involved in about 50 per cent of all cases, and the parts involved, in order of frequency, are: buccal mucosa, palate, gums, posterior pharyngeal wall, larynx, vagina, prepuce, conjunctiva, rectum and esophagus (Becker and Obermayer [5]).

I had the privilege of making a laryngeal examination in one of the late Dr. W. H. Mook's cases a few years ago and preserved the following record of my findings: "Groups of slate-colored or darker elevations are scattered about the laryngeal mucosa. Distributed over the rima glottidis and aryepiglottic folds are some lesions having the appearance of the well known tattooing from embedded cinders or gun powder grains without visible elevation, while others definitely protrude above the surrounding mucosa resembling partially submerged dark splinters or lead pencil points broken off slightly above the surface. Similar lesions are scattered promiscuously over nearly all the visible oral and pharyngeal mucous membranes, including the faucial pillars."

KERATOSIS FOLLICULARIS (DARIER'S DISEASE)

Brief reference has been made already to this disease and some of the characteristics of its cutaneous lesions. It is a rare chronic disease of the skin. The early lesions are described as predominantly follicular, i.e., a keratosis about the mouths of follicles, but some authors have stated that the majority of the early lesions are interfollicular. These primary lesions are small, slightly elevated, yellowish to dark gray or dark brown, conical papules, from 1 to 3 mm. in diameter. If a semiglobular keratotic apical plug can be removed, a funnel-shaped crater remains, often containing a hair. Occasionally an individual lesion is flat and dark red with no central crater and may resemble a lichen planus papule. When the disease progresses, the papules coalesce, becoming interfollicular; they were described by Stelwagon as not unlike a nutmeg grater. Persons with the disease frequently emit a peculiar offensive odor.

The regions of the body that perspire most freely are chosen by these lesions as their sites of predilection, but excessively hairy areas are also commonly selected.

Fabry and others have called attention to involvement of the oral mucosa in the form of isolated nodules on the tongue, the buccal mucosa or the hard and soft palate. One case report [6] of typical lesions in the esophagus was also found.

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5. Becker, S. W., and Obermayer, M. E.: Modern Dermatology and Syphilology, Philadelphia, J. B. Lippincott Company, 1940.

6. Mercadal Peyri, J.; Valls, A., and Dulanto, F.: Un caso de coexistencia de psoriasis y enfermedad de Darier con lesiones típicas de ésta última en las mucosas esofágica y rectal, Actas dermo-sif. **32**:608-616 (April) 1941.

ERYTHEMA MULTIFORME

This acute inflammatory disease is characterized by a cutaneous eruption of erythematous raised areas of multiferous kinds and shapes, varying from simple papules to fantastic figures with raised borders and sometimes depressed centers. As the lesions spread peripherally, their color may change to various shades of purple or bluish red, and when irregular circles are formed the types known as erythema annulare and erythema iris may result. Later new lesions may begin near the centers of the older ones, and by repetition of this process a series of concentric rings appear. Coalescing of the margins of proximal lesions may produce larger scalloped-edged areas.

Symmetric, painful or tender erythematous nodules on the shins and extensor surfaces of the lower limbs which do not ulcerate or suppurate are characteristic of erythema nodosum. This is considered a separate clinical entity and not a form of erythema multiforme, but the two conditions do occur together at times.

The prognosis is good except in the persistently chronic or recurrent forms, and a selective distribution of lesions about the knees and large joints indicates a serious condition with none too good an outlook.

The mucosal lesions are similar to those of the skin except that bullous forms predominate and annular, circinate or iris-shaped lesions are not so pronounced. Elevated infiltrated areas resembling erythema nodosum have been described, and I feel reasonably sure that recently I encountered such a case in which the larynx was involved.

The patient was a white man 50 years of age on whose legs an eruption had developed five days after the administration of sulfadiazine (2-[paraaminobenzene-sulfonamido]-pyrimidine) had been discontinued. Sixty grains (4 Gm.) daily had been administered for eight days in the treatment of pneumonitis following a severe virus infection of the respiratory tract (influenza). Within twenty-four hours the eruption had spread to the thighs, arms and forearms. The next day Dr. C. W. Lane (a dermatologist) saw the patient in consultation and reported a combination of erythema multiforme and erythema nodosum, but he could not say whether the cause was accumulation of the drug from poor elimination or severe toxemia of the disease.

An otolaryngologic consultation was finally requested because of hoarseness. This symptom was first noticed about the time of the outbreak of the eruption, which was seven days old and fading when I examined the larynx.

The upper surfaces of both vocal cords were irregularly lumpy because of dark or dull red nodes apparently too firm to be due to edema alone. The anterior half of each cord was most prominently involved. On the left cord near the anterior commissure was a node shaped like a loaf of bread overlapping a similar lesion on the right cord, which when revealed to inspection showed a white longitudinal ridge of compression evidently produced by being pinched between the two cords in phonation.

These nodes gradually became smaller (with voice rest) and had practically disappeared in about seventeen days. The cutaneous lesions disappeared several days before the larynx was clear.

In spite of the fact that the administration of sulfadiazine had been discontinued so long before the cutaneous eruption appeared, I believe the eruption was a drug reaction; a few days before I saw this patient, my 9 year old son had broken out with a typical generalized erythema multiforme eruption of the skin just five days after a pediatrician had discontinued administration of the same drug.

DERMATITIS HERPETIFORMIS (DUHRING'S DISEASE)

This disease is a chronic recurrent disorder characterized by intensely itchy or burning groups of erythematous papules, vesicles, bullae or pustules in highstrung, nervous, undernourished persons. Sites of predilection of the eruption are the scapular regions, the sacrum, the lateral surfaces of the thighs and the extensor surfaces of the upper arms or forearms, and the distribution is usually symmetric. Vesicular forms resemble herpes simplex and the bullous forms pemphigus. The bleb tents are so tough that they seldom rupture spontaneously but are usually opened by scratching.

The type of lesion may change perceptibly in successive outbreaks, and multiformity may be noted even during the same wave.

The mucous membranes of the mouth, nose, esophagus or genitalia may be involved, but only in rare instances, and when they are the lesions are of the vesicular or bullous type. The latter type may be confused with pemphigus, which it closely resembles. Pemphigus, however, does not include grouped vesicles; the bullae are more uniform in size, and, while often painful, they are less itchy.

PEMPHIGUS

Acute and chronic forms of pemphigus are recognized, and the acute form is practically always ushered in by severe prodromal symptoms or chills and high fever of a septic type, soon followed by a cutaneous eruption; this may begin as erythematous macular lesions, but they rapidly develop into bullae from 1 to 10 cm. in diameter. The serous content of these bullae usually is clear, but it may be hemorrhagic. Either type usually arises from an apparently normal skin, or else the lesions are surrounded by only a slight pinkish or faint red areola.

Vaccination or infected wounds may precede the onset of the acute form. When staphylococci, streptococci, pneumococci or colon bacilli are recovered from the blood stream or bullae—and sometimes from both—with a leukocytosis and often an eosinophilia, a severe septicemia is likely present, and, if so, recurring crops of fresh lesions may rapidly follow one another until death finally occurs.

The chronic form may also recur or persist until a fatal termination results. The lesions do not rupture spontaneously unless severely trau-

matized, but their usually clear initial serum eventually becomes turbid and the blebs become less tense. When ruptured by injury the denuded oozing surface may become covered with dark brown scabs or crusts. After epithelization and complete healing take place no permanent scar is left.

The characteristic lack of adhesion between the stratum corneum and subjacent prickle layer of the epidermis may be demonstrated by the ease with which abrasions are produced by delicate trauma on apparently unaffected areas between bullae. Another demonstration of the same characteristic is known as Nikolsky's sign; this depends on the fact that a pemphigus bleb can be moved within the epidermis from a diseased area to an adjacent apparently normal one without rupture by careful finger manipulation. This was once thought to be a highly diagnostic sign of pemphigus, until it was discovered to be present also in other diseases.

The mucosal lesions of pemphigus are seldom found as intact blebs except when freshly formed, because they rupture so early and easily. The maceration from moisture in the mouth makes the tentlike coverings of epithelium very delicate and fragile. The denuded areas may be rather extensive and scalloped by coalescence of spreading lesions, and they sometimes ulcerate or become extremely foul and offensive. As with so many other mucosal lesions, the diagnosis of pemphigus blebs on mucous membranes may depend on the accurate identification of an accompanying eruption on the skin.

EPIDERMOLYSIS BULLOSA

This disease is characterized by the formation of bullae at sites of trauma. Two types of the disease are described: One is designated as epidermolysis bullosa simplex and the other, a more destructive variety, as epidermolysis bullosa dystrophica.

The lesions are usually bullae, although often nothing can be recognized but raw areas where the epithelium has been easily rubbed off by trauma or mechanical irritation. The bullae form between the dermis and the epidermis; so when raw areas appear only the latter layer of tissue is missing.

Apparently any area of skin or mucosa may be affected, but those most frequently involved are the ones most likely to be traumatized, such as the hands, elbows, heels, feet and knees.

In the dystrophic type, the bullae are commonly hemorrhagic and when healed may leave depigmented or hyperpigmented atrophic areas. Trophic changes of the nails are frequent. The mucosal lesions are more common in the dystrophic type and are also more severe, persistent and often actually ulcerated. As is the usual rule with mucosal

bullae, intact mature lesions are seldom seen, as they rupture too easily, leaving raw red tender areas with only shreds of the epithelial covering clinging to their periphery or retaining a thin covering of a fibrinous pellicle.

SCLERODERMA

Scleroderma is a chronic disease characterized by a circumscribed or a more or less diffuse hardening of the integument, a condition described by Stelwagon as "hidebound." The involved areas are usually pigmented or variously tinted, indurated spots, streaks, bands, patches or diffuse lesions, often stiffened to rigidity. The neck is most frequently the primary site of predilection, but the face and almost any other region may be attacked and the involvement eventually becomes rather extensive and irregular. Underlying muscles may undergo a certain amount of atrophy, and in the limbs this may be noted even where the overlying skin is not visibly affected. Three stages of the disease are recognized, i.e., edematous, indurative and atrophic.

Circumscribed scleroderma is known as morphea, but Duhring contended that the two varieties are not identical pathologic processes. Other authors have cited cases in which both varieties have been observed in typical form and simultaneously in the same person, and also cases have been reported in which one form has merged into the other.

Several authors state that scleroderma may involve mucous membranes around the nose and mouth showing changes similar to those of the skin and appearing in acute and chronic, circumscribed and diffuse forms. Atrophy of the mucous membrane with adhesion to the submucous tissue eventually develops. It reaches the mouth through the choanae, producing infiltrated areas covered with a somewhat bluish red mucosa. Later, hard nodules appear, and they are somewhat tender on pressure and may ulcerate. As in the skin, mucosal scleroderma undergoes three periods (edema, induration and atrophy), the last of which eventually persists.

Stelwagon [4] mentioned involvement of the oral mucous membrane and stated that this has been observed "even when the integumentary involvement was limited." He also cited Dercum's statement that the teeth may loosen and fall out.

POROKERATOSIS (MIBELLI)

This rare chronic disease is believed to be predominantly familial. The fully developed lesions are highly characteristic and consist of roughly oval or rectangular plaques with a somewhat atrophic center and an elevated border surmounted by a groove or furrow. Originating from this groove a keratotic ridge is described. Cross sections of the border are triangular. The early individual cutaneous lesions begin at

the mouths of glands, from the name of which peculiarity the prefix poro is derived.

When the mucous membranes are involved the lesions are described as 1 cm. or less in diameter. The lesions lack the usual mucosal glitter and are round or oval, white and finely granular, surrounded by a border having the appearance of a fine thread embedded just under the surface. Minute examination may reveal a small furrow at the summit of this threadlike border.

OTHER DISEASES

In addition to those diseases that are commonly considered as predominantly dermatologic, the following conditions also present mucosal or dermatologic manifestations:

Cheilitis—Inflammation of the lips.

Cheilitis Exfoliativa—A chronic form usually showing silvery scaling, which is sometimes mistaken for that of other diseases; usually accompanied by seborrheic skin.

Fordyce Disease—Small yellowish submucous oval or rounded areas, usually on the buccal mucous membrane; harmless unless discovered accidentally by an introspective patient in whom a cancer phobia develops.

Stomatitis—Inflammation of the mouth.

Stomatitis Aphthosa—Canker sores or small superficial ulcers of the oral mucous membranes.

Mucosal Keloids—Seldom acknowledged or recognized but in rare instances as real and pronounced as cutaneous keloids.

Mucosal Argyria and Localized Facial Argyria—Often produced by prolonged use in the nose or pharynx of colloidal silver salts for prophylaxis.

Leukoplakia—White keratosis of the mucous membranes from some form of irritation; potentially malignant; diagnosis often difficult.

Certain Diseases of the Tongue—

Congenital fissured tongue, often called scrotal tongue. Transitory benign plaques of the tongue: Glossitis areata exfoliativa and geographical tongue are apparently different phases of the same process.

Lingua nigra (black tongue) pigmented hairy coating due to hyperkeratosis of papillae, dyes or a fungous infection.

Glossodynia with an objectively normal tongue, invariably a neurosis pure and simple and often incited by some phobia (cancer or tuberculosis usually).

Moeller's glossitis, characterized by irregular, glazed, fiery red, slick-appearing areas on the dorsum of the tongue near the borders and surrounding the tip; often accompanied by anemia; no ulceration; often confused with transitory benign plaques.

Oral and Gingival Manifestations of Heavy Metal Supersaturation —Mercurialism (salivation) and lead and bismuth poisoning.

Mucosal Drug Reactions—Caused by various drugs and often extremely confusing but seldom typical of anything else.

Mucosal Feigned (factitial) Lesions—Usually can be recognized by their artificiality, i.e., always exaggerated and too fantastic to be real.

Agranulocytosis—I have never encountered 2 cases in which the mucosal lesions were exactly alike. I still wonder in how many the condition is due to barbiturates and other drugs. The leukopenia is constant.

Hemiangioma and Other Types of Angioma.

Verrucous and Papillomatous Lesions—Also various other benign and malignant mucosal lesions of the respiratory tract, with or without accompanying cutaneous or other extensions.

Mucosal Edema and Allergic Manifestations—Lesion similar to the cutaneous manifestations of urticaria and angioneurotic edema.

Avitaminosis—Various newer classifications of certain diseases now believed to be due to vitamin deficiency.

Pellagra—Characteristic glossitis (a smooth fiery red tongue) plus gastrointestinal and mental symptoms, along with the brown scuffy neck and wrist band or cuff.

Rhinoscleroma—A neoplastic condition not to be confused with rhinophyma, which is not; a much softer type of hyperplasia (the third degree of acne rosacea, now called rosacea).

Osler's Disease, or Multiple Hereditary Telangiectasia—Patients with this disease frequently come to the rhinologist for control of persistent epistaxis.

Thrombopenia—Patients with this disease, with its petechiae, some of which may rupture and bleed spontaneously, also come to the rhinologist for control of epistaxis.

Granulomas—

Granuloma pyogenicum
Tuberculous granuloma
Lupus granuloma
Syphilitic granuloma
Leprous granuloma
Blastomycosis
Granuloma venereum

Moniliasis Candida and Other Mycotic Diseases—including thrush and sprue.

Rocky Mountain Spotted Fever and Other General Diseases with Combined Cutaneous and Mucosal Lesions from Insect Bites—Rocky mountain spotted fever, due to the bite of the tick (Dermacentor occidentalis), is a serious disease, often fatal. The cutaneous eruptions (petechiae, purpura and erythema) follow a chill and high fever with severe systemic reactions. Headache and epistaxis occur in the second week, and late in the disease the mouth and throat are involved and hemorrhages occur from the entire gastrointestinal tract with effusion into the joints.

Foot and Mouth Disease—A contagious malady of cloven-footed animals occasionally affecting man, when it is characterized by fever and a vesicular eruption of the mucous membranes of the mouth and pharynx; the skin of the hands and feet may also become involved. The disease usually terminates in recovery within a few days.

Xanthomatosis—Now known to produce both cutaneous and mucosal manifestations and several syndromes, some of which are serious.

COMMENT

The enanthematous lesions of exanthematous diseases are purposely not included in this paper because it is assumed that most of the otolaryngologists can hold their own fairly well with the dermatologists, pediatricians and internists in recognizing such lesions as Koplik spots in measles and strawberry tongue and erythematous throats in scarlet fever.

Other phases of this subject must be omitted in a paper of this sort. A few paragraphs could be devoted to cutaneous eruptions appearing on or about the nose, mouth and ears. Some of these are caused by staphylococci or some other irritant in the discharges from these orifices, e. g., infectious eczematoid dermatitis, impetigo, furunculosis and erysipelas, while other disorders, such as regional adenopathy, are frequently brought to the attention of the laryngologist because a deep etiologic connection is suspected, when the disorder is just as often caused by some overlooked cutaneous inflammation. Finally, it is assumed that numerous other diseases, such as Vincent's angina, mucosal herpes simplex and herpes zoster of the respiratory tract, are so familiar to otolaryngologists that they would not be interested in their discussion here.

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SCLEROMA

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HISTORY

Rhinoscleroma was so named by von Hebra, a Viennese dermatologist, who, with his colleague Kaposi, first described the disease. His first cases, 9 in number, 4 patients males and 5 females, were presented before the medical society of Vienna in 1870; but as early as 1852 he had observed a case in Paris which in retrospect he believed to be an instance of the same condition. Thirty years prior to von Hebra's presentation of these cases, a Dr. Bierkowski, of the surgical clinic of Cracovie, had made plaster molds which showed all the characteristics of rhinoscleroma. He considered this disease to be some bizarre form of cutaneous cancer and consequently made no effort to classify it as a new disease entity. Von Hebra and Kaposi (1874) and Bierkowski were also ignorant of the true nature of the disease, for they believed it to be a type of granulation sarcoma, this supposition being at sharp variance with the prevailing one of the time, which considered it a peculiar form of syphilis. This erroneous idea persisted until Geber, von Hebra's assistant, noting the perivascular cellular infiltration and connective tissue proliferation in histologic preparations, propounded the hypothesis that rhinoscleroma is a specific chronic inflammation of unknown causation. Mikulicz, in 1876, working in Bilroth's clinic, added weight to this theory with his excellent histopathologic studies, in which he described for the first time the large foam cells which today bear his name. It remained for von Frisch to discover, in 1882, a diplobacillus occurring in the nasal secretions and affected tissues of rhinoscleromatous patients. He, as well as others, felt this to be the etiologic agent, and, indeed, there are those of the present day who are of the same opinion. It must be said, however, in this regard, that Koch's postulates have never been fulfilled, and there is considerable doubt as to whether the disease has ever been reproduced in lower animals.

Read before the Section on Laryngology, Otology and Rhinology at the Ninety-Third Annual Session of the American Medical Association, Atlantic City, N. J., June 10, 1942.

Laryngeal scleroma was described by Gerhardt in 1876. With this work came the realization that scleroma was not a disease confined strictly to the nasal cavity, as the term rhinoscleroma would indicate. The more appropriate term scleroma was suggested and in 1932 was adopted officially by the International Congress of Otology, Rhinology and Laryngology at Madrid, Spain. At this congress, moreover, a comprehensive survey of the disease was made. The *Rapports* constitute the most accurate source of scleroma statistics. From these *Rapports* much of the data, historical and otherwise, in this paper has been gleaned.

INCIDENCE

Scleroma, though once thought to exist only in central and southeastern Europe, has been found to be neither geographically nor ethnically limited. It is true that most cases have occurred in the central and southeastern European countries, where the disease is endemic, but with the widespread immigration of these peoples to other parts of the world, scleroma is now ubiquitous and appears to be increasing steadily in nations of the Western Hemisphere, and more particularly in those of South and Central America.

In the United States and Canada there have been a total of 102 patients reported on, of whom 16 have been native born. These figures have been arrived at by a careful review of the literature and by communicating whenever possible with the authors who have reported cases. The latter step was found to be essential, for in some instances the same cases have been reported by more than 1 person. Included in this number are also the cases reported in this paper.

Table 1 summarizes the pertinent data on the foreign-born patients, giving whenever possible the birthplace, age, sex and anatomic involvement. In many instances such data are not to be had and cannot therefore be tabulated. Table 2 lists the native-born patients with similar data.

It might be of interest to add that there are several who, besides being born in this country, spent their life in and around the immediate vicinity of their birthplace and were never exposed to scleroma so far as they could remember. Jervey, of Greenville, S. C., stated that the 2 cases which he had observed had occurred in native-born residents of South Carolina and that he believed an endemic focus of scleroma exists around Spartanburg, S. C.

Belinoff's statistics on scleroma given at the International Congress are the most accurate and comprehensive extant. Two thousand, three hundred and sixty-one cases were reported. In these, 973, or 37 per cent,

of the patients were males and 1,426, or 54 per cent, were females. The sex of 237 patients was not indicated. If these cases are discounted and the percentages are then computed, the following figures are arrived at: females 58.5 per cent and males 41.5 per cent, the females thus preponderating by some 17 per cent.

So far as age incidence is concerned the following figures are informative (the age was not given in 18 per cent of the cases):

Age Group, Years	Percentage of Patients
1 to 15	5
16 to 25	39
26 to 35	20
36 to 45	11
46 to 55	4
Above 56	3

As for the economic level, Belinoff's figures are significant:

Classification	Percentage of Patients
Workers and peasants	94.5
Intellectuals	1.3
Employees, so-called white collar workers	4.2

It is obvious that scleroma is a disease of the lower stratum of society and the greatest number of cases occur during the most productive decades, viz. 16 to 35 years.

SYMPTOMS AND CLINICAL COURSE

Scleroma is a disease characterized by an insidious onset, its symptoms being referable to the pathologic changes that occur in the respiratory passages. Not infrequently a patient may complain early of nasal crusting or may notice a foul-smelling discharge. Later, as the disease progresses blockage of the air passages occur, due to the extension of the disease, particularly in the region of the inferior meatus. Epistaxis is common. Further progress may lead to distortion of the nasal architecture to such a degree as to render the affected person almost unrecognizable. In addition, the disease process may spread to involve the skin of the upper lip, cheeks, gums, antrum, lacrimal ducts, eustachian tubes, nasopharynx, soft and hard palate, tonsils, pharynx, larynx, trachea and bronchi, even to second and third order. With involvement of any of these structures impairment of function follows, giving rise to such symptoms as hoarseness, brassy cough, difficult and stertorous breathing, ozena, deafness, tinnitus and sinus headaches. When laryngeal stenosis becomes pronounced, as frequently is the case, tracheotomy becomes necessary to prolong life.

The constitutional symptoms so far as the disease process is concerned are practically nil. Mechanical blockage of air passages resulting in a dry oral cavity leads to ageusia and concomitant anorexia. Consequently, it is not unusual to find scleromatous patients ill nourished and cachectic, particularly in the late stages of the disease.

In most cases of scleroma the process begins in the nose and for long periods may involve only that organ. From here it spreads to involve other structures, such as those previously enumerated. Primary foci do occur elsewhere, but rarely. Belinoff's statistics in regard to localization of the disease are as follows:

Location	Cases	
	Number	Percentage
Nose	1,336	60
Larynx	1,332	60
Nasopharynx	613	28
Pharynx	368	16
Trachea and Bronchi	196	9

In 430 cases insufficient data were given to warrant their being statistically analyzed.

As with most chronic diseases, scleroma pursues a lengthy course extending over a period of decades. Death results from intercurrent infection or asphyxia unless therapeutically effective measures arrest the process.

PATHOLOGIC CHANGES, GROSS AND MICROSCOPIC

Szmurlo divided scleromatous lesions into two types: The diffuse type presents a more or less uniform thickening or thinning of the skin and mucous membrane, while the discrete, or circumscribed, type consists of nodules or tubercles scattered over the affected surface. Rarely does one see a pure form of the disease; usually there is a mixture of the two types. The initial stage of the diffuse type strongly resembles atrophic rhinitis, as the mucous membranes become thin, dry and covered with large yellowish crusts. Later, when infiltration becomes more pronounced, the tissues thicken and assume a hard cartilaginous appearance. At times the diffuse type assumes an almost invasive quality; in such instances the architecture of the affected organ is seriously disturbed, resulting in ghastly deformities. The infiltrative type, though limited not infrequently to the nose, usually spreads downward to involve the soft palate, nasopharynx, pharynx and larynx, the pathologic changes in each case being essentially of the same character. As the disease progresses the scleromatous tissue tends to contract. This further distorts the structure involved and reduces function considerably. The lesions of the nodular, or tubercular, type begin as circumscribed nodules each the size of a grain of wheat and of a reddish violet hue

TABLE 1.—*Scleroma in Foreign-Born Patients*

Case	Author	Date	Sex	Age	Birthplace	Site of Lesion
1	Jackson	1893	F	54	Hungary	Nose
	Pollitzer	1894	..	..	.....	.....
2	Freudenthal	1896	M	45	Galicia	Nose, pharynx, larynx
3	Freeman	1900	M	34	Russia	Nose
4	Jarecky	1901	F	32	Russia	Nose, pharynx, larynx, trachea
5	*Fisher	1902	..	..	Austria	.....
6	*MacPherson	1902	..	..	Russia	.....
7	Danziger	1905	F	..	Austria	Nose
8	Danziger	....	F	50	Austria	Nose, soft palate, trachea
9	Toeplitz and Kreyder	1905	F	25	Galicia	Mouth, nose
10	Toeplitz and Kreyder	....	..	..	.....	.....
11	Day	1906	F	27	Galicia	Mouth, nose
	Kenyon	1906	M	26	Bohemia	.....
12	Pollitzer	1906	F	37	Austria	Nose
13	Gotthell	1906	F	30	Russia	Nose, upper jaw
14	Lustgarten	1907	F	18	.....	Larynx
15	Stubbs	1907	F	21	Poland	Nose, larynx
16	Ballin	1907	F	53	Poland	Nose, pharynx
17	Perkins	1907	M	35	Austria	.....
18	Mayer	1907	F	16	Poland	Trachea
19	Mayer	1907	F	23	Poland	Larynx, nose
20	Mayer	1908	M	18	.....	Nose, larynx
21	Mayer	....	F	9	.....	Nose
22	Mayer	....	F	21	Poland	Nose, larynx
23	*Moll	1908	..	..	Poland	.....
24	Gotthell	1908	F	70	Russia	.....
25	Gotthell	....	F	29	Russia	.....
26	Guntzer	1909	M	19	Russia	Nose
27	Smith	1912	F	25	Poland	Nose, pharynx
28	*Doerann	1910	..	..	Poland	.....
29	*Levy	1910	..	..	Russia	.....
30	Braun	1910	M	27	Italy	Nose
31	Thro	1910	M	35	Russia	Nose
32	Durkee	1910	F	20	Russia	Nose, larynx
33	*Chambers	1911	..	..	Hungary	.....
34	Iglauer	1912	F	28	Russia	Nose, pharynx
35	Smith	1912	M	30	Poland	Nose
36	Smith	....	M	18	Russia	Nose
37	*Schell	1914	..	..	Russia	.....
38	Alderson	1914	M	24	Mexico	Nose
39	Kaempfer	1914	F	19	Russia	Nose
40	Guttman	1915	F	26	Hungary	Nose, larynx (?)
41	Holinger	1915	..	..	Ruthenia	Nose
42	*Parkinson	1916	..	..	Ireland	.....
43	Gotthell	1917	M	24	Mexico	Nose
44	Gotthell	....	M	22	Austria	Nose
45	Stelwagon	1918	F	52	.....	Nose
46	Bailey	1919	M	33	Austria	Nose
	McGee and Wise	1917	..	..	.....	.....
47	*Grant	1920	..	..	Russia	.....
48	*Douglas	1920	..	..	Germany	.....
49	*Douglas	1922	..	..	Russia	.....
50	*Nilsen	1922	..	..	Russia	.....
51	*Palmer	1922	..	..	Galicia	.....
52	*Conway	1924	..	..	Italy	.....
53	*Sigmann	1925	..	..	Austria	.....
54	*Flynn	1927	..	..	Poland	.....
55	*Myerson	1928	..	..	Russia	.....
56	Figi and Thompson	1928	M	32	Austria	Nose, pharynx, larynx
57	Figi and Thompson	....	M	27	Russia	Nose, pharynx
58	Figi and Thompson	....	M	27	Roumania	Nose, pharynx
59	Figi and Thompson	....	M	35	Roumania	Nose, pharynx, larynx
60	Figi and Thompson	....	M	30	Galicia	Nose, pharynx, larynx
61	Crowe	1928	M	30	Poland	Pharynx, larynx
62	Crowe	....	M	35	Italy	Larynx
63	*Thomas	1929	..	..	Germany	.....
64	*Flynn	1929	..	..	Poland	.....
65	*Lyon	1930	..	..	Poland	.....

* Jackson's cases compiled for the Madrid Congress from answers to questionnaires sent to two thousand otolaryngologists in the United States.

TABLE 1.—*Scleroma in Foreign-Born Patients—Continued*

Case	Author	Date	Sex	Age	Birthplace	Site of Lesion
66	*Hadje	1930	..	..	Hungary	.....
67	Alderson	1932	M	30	San Salvador	Nose, pharynx, larynx
68	Lewis	1932	..	24	Mexico	Nose, pharynx, larynx
69	Lewis	....	..	29	Mexico	Nose, larynx, soft palate
70	Schwartz	1935	M	33	Russia	Nose, larynx, soft palate
71	Schwartz	....	F	42	Russia	Nose, larynx, trachea
72	Bingham and Cutler	1935	M	23	Mexico	Nose
73	Chamberlin	1936	M	24	Russia	Larynx only
74	Chamberlin	....	M	52	Russia	Nose, larynx
75	Ireland	1936	M	24	Poland	Nose, larynx, pharynx
76	Morrison	1938	F	63	Mexico	Nose, pharynx, right ear
77	Morrison	....	M	28	San Salvador	Nose, antrums
78	Morrison	....	..	26	Guatemala	Nose, larynx
79	Gadomski	1939	F	26	Poland	Nose, pharynx
80	Simpson and Ellis	1939	F	55	England	Nose
81	Weiss	1939	F	53	Mexico	Nose
82	Weiss	....	F	60	Poland	Nose
83	Cunning	1942	M	53	Austria	Nose, pharynx, larynx
84	Cunning	1942	F	47	Russia	Nose, larynx, trachea
85	Cunning	1942	M	56	Italy	Larynx
86	Cunning	1942	M	62	Italy	Larynx

TABLE 2.—*Scleroma in American-Born Patients*

Case	Author	Date	Sex	Age	Birthplace	Site of Lesion
1	*Tarrell	1890	..	..	New York	.....
2	Wende	1895	M	11	Buffalo	Nose
	Roe	1896	..	..	.....	.....
3	*Moore	1911	..	..	Arkansas	.....
4	Lieberthal	1911	F	26	.....	Nose, pharynx, lip
5	*Alloway	1912	..	..	North Carolina	.....
6	*Alloway	1920	..	..	Cincinnati	.....
7	Watkins	1921	M	23	Baltimore	Larynx
8	Wood	1925	M	24	Trenton, N. J.	Nose, larynx, pharynx
	Figi and Thompson	1928	..	..	.....	.....
9	*Howland	1925	..	..	Michigan	.....
10	*Jervey	....	..	..	Spartanburg, S. C.	.....
11	Canfield	1933	F	17	Michigan	Nose, larynx, pharynx
12	Helwig and Jaime	1933	M	20	Kansas	Larynx
13	Stout	1934	M	25	Arkansas	Nose
14	Alderson	1937	F	29	California	Nose, hard palate, lip
15	Simpson and Ellis	....	F	41	Greenville, S. C.	Nose
	*Jervey	....	..	..	.....	.....
16	Cunning	1942	M	21	Perth Amboy, N. J.	Nose, pharynx, larynx
17	Cunning	1942	M	34	Brooklyn	Nose, pharynx, larynx

* Jackson's cases compiled for the Madrid Congress from answers to questionnaires sent to two thousand otolaryngologists in the United States.

and edematous consistency. They grow slowly and eventually coalesce to form larger lesions which progressively block the affected region. With both types of lesions the ultimate result is the same, contraction of the scleromatous tissue into thick gray atrophic scars resulting in marked deformation of structure.

The disease is limited to the skin and mucous membranes in all cases, cartilage, bone, lymph glands and muscle being spared. Pressure necrosis may occur, but frank ulceration is the exception and not the rule.

Microscopically, scleroma presents a fairly characteristic picture. A stained section of scleromatous tissue will, depending on the stage of the disease, present in varying numbers the large vacuolated foam cell of Mikulicz with a diameter of 100 to 200 microns. These cells are believed to be degenerated connective tissue or reticuloendothelial cells, and it is within them that von Frisch bacilli are usually found. These foam cells are swollen (hydropic), their nuclei either appearing indistinctly or disappearing completely. In the less swollen cells the nuclei remain distinct and the protoplasm preserves its granular character.

Mikulicz cells, however, are not pathognomonic for scleroma, as they are found in some other conditions, such as leprosy, glanders, bubonic plague, granulation tissue of wounds, inflammation edema and inflammation of sweat glands.

The hyaline cells of Russell or Unna are also found in scleroma lesions. These are supposed to represent degenerated plasma cells, and with routine hematoxylin and eosin stains the cytoplasm becomes brilliant pink, while the cells are elliptic in shape with the nucleus situated in one pole. Such cells, while more numerous in scleroma, are also found in tuberculosis, syphilis, leprosy, chronic granulomas and various malignant tumors and filtrable virus diseases.

In scleroma the epithelium presents extreme papillary hyperplasia and may show pearls, which in many cases caused early workers to make an erroneous diagnosis of epithelioma. This is, however, a pseudo-epitheliomatous lesion.

The typical pathologic picture is that of a chronic inflammatory process in which considerable lymphocytic and plasma cells have occurred and in which also the Mikulicz and the Unna cells are to be found, the former harboring within their walls von Frisch bacilli.

ETIOLOGY

The etiology of scleroma is still an enigma. The von Frisch bacillus, which can be cultured from all scleromatous lesions, is said by some to be the causal agent. It is difficult, if not impossible, to distinguish this organism from other members of the capsulated group, and since organisms of a like nature are frequently found in the nasal secretions of healthy persons the von Frisch bacillus cannot be definitely incriminated. Scleroma has never been reproduced in lower animals; many attempts to carry out such a procedure have failed. Scleroma is generally conceded to be an infectious disease, but it seems to be slightly contagious. Tomasek, Quast, and more recently Kouwenaar, Massland and Wolff and Belinoff (1936) claimed to have developed a complement fixation test which is of questionable value in diagnosis. Topley and Wilson admirably summed up the case against the von Frisch

bacillus in stating that "the probability is that they are mere secondary invaders which grow freely in the nose of patients suffering from the disease."

Believing that scleroma might be caused by a virus, or perhaps a virus and the von Frisch bacillus acting symbiotically, I have carefully examined sections for inclusion bodies, but all strides thus far have been fruitless. In the near future it is expected that work designed to prove or disprove this hypothesis will be carried out.

TREATMENT

The great number of therapeutic agents advocated for the treatment of scleroma is evidence that no effective method of dealing with the disease has yet been found. Intravenous injection of arsenicals, mercurial inunctions, oral administration of iodides, local application of escharotics, vaccinotherapy, surgical intervention and electrocoagulation, as well as numerous other remedies, have all been tried singly and in combinations, but with poor results.

Roentgen therapy, first employed by Gottstein, has been found to offer the most promising method of treatment, particularly in the early stages. When the disease has become firmly established and has spread to involve the subglottic regions, such therapy is necessarily limited because of the contiguous vital structures.

The use of radium has also been found to be of value for certain early lesions involving only small areas. Cures are said to have been obtained with roentgen and radium therapy, but, in general, the feeling of radiologists is that such therapy is palliative at best and must be repeated as recurrence of the disease dictates. Weiss, in a comprehensive article, gave the salient features in regard to teleradium therapy as well as the histologic tissue changes that follow treatment of this kind.

In 3 of my cases (cases 1, 2 and 3) sulfanilamide was tried, and in 2 cases sulfadiazine. Proper therapeutic blood levels were maintained for as long intervals as the patient could tolerate the drug. Several courses were carried out with the patient hospitalized. In each instance immediate general improvement took place, and after two weeks of intense therapy examination of pathologic specimens indicated a slight but definite improvement. Von Frisch bacilli, though still present, diminished in number. In each case with the cessation of therapy the condition within one month returned to the same status as that prior to beginning treatment, and in 1 instance it became worse. I am therefore inclined to believe that sulfanilamide and allied drugs serve to control the secondary invaders and have little or no effect on the actual scleromatous lesions.

DIAGNOSIS

Diagnosis can be established only by biopsy. Culture of von Frisch bacilli is of no importance, and the complement fixation test has yet to prove its worth.

In most cases of scleroma the diagnosis is missed because the disease is not suspected. It is not infrequently confused with such conditions as syphilis, tuberculosis, carcinoma and other new growths as well as various granulomas. If the disease is kept in mind and a specimen for biopsy taken, diagnosis is but a simple matter.

REPORT OF CASES

The following 6 cases came under my observation at the bronchoesophagologic clinic of the Manhattan Eye, Ear and Throat Hospital during the past two years.

CASE 1.—A 46 year old, married, rather obese white woman of Russian birth was admitted to the hospital on Aug. 29, 1938 for the first time. She then complained of progressive hoarseness and dyspnea of four years' duration. Two months prior to admission an emergency tracheotomy had been carried out at the Kingston Avenue Hospital, Brooklyn, and on admission the tube was still in place. Her family history and past personal history were noncontributory.

Physical examination revealed no essential abnormalities except in the larynx. Her cords were found to be moderately thickened and infected, while the false cords were markedly swollen and the posterior commissure filled with redundant tissue. A specimen for biopsy taken from the posterior commissure and false cords revealed only chronic inflammatory tissue. Repeated biopsies failed to reveal any other diagnosis, and the patient was discharged on October 8. During hospitalization her Wassermann and Kahn reactions were repeatedly negative and other laboratory findings were not of interest. Roentgen examination of her chest revealed no pathologic changes.

The patient was readmitted about two weeks later with laryngotracheal bronchitis, which responded to routine therapy.

She was readmitted on March 21, 1939 for removal of her tracheotomy tube and plastic closure of the wound.

From that time until her next admission, Jan. 8, 1941, she was followed in the bronchoesophagologic clinic, where biopsy of tissue from both the larynx and the nose showed scleroma. During this time her condition had become much worse, and on admission her cords were found to be slightly thickened and infected, with marked enlargement of the right false cord, which appeared covered with granulations. A large granular subglottic mass could be seen just below the posterior commissure. The posterior pharyngeal wall appeared very dry and glazed, and both inferior turbinates were atrophic with marked crusting. Physical examination otherwise showed nothing abnormal.

Sulfanilamide therapy was begun at once, and the concentration in the blood was maintained around 10 mg. per hundred cubic centimeters. Her temperature immediately began to rise and by the eighth day reached 104 F., subsiding to normal by the twelfth day. No cause for this fever could be determined, although several roentgenograms of the chest were taken. The patient was left on sulfanilamide therapy for twelve days and then discharged. Her course, except for the develop-

ment of a slight cough and for the fever, was uneventful. When she left her cords appeared normal, the swelling of her right false cord had considerably subsided and the subglottic mass had definitely decreased in size. Subjectively the patient felt much improved.

Her improvement continued for two to three weeks and then began to regress. By the time of her next admission, March 12, her condition was practically the same as it was on the last admission. Sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) therapy was begun and the blood concentration was maintained around 5 mg. per hundred cubic centimeters for fourteen days, when the patient was discharged as unimproved.

Since that time the patient has been followed in the clinic. The disease process has spread down and into the right bronchus. In addition, the laryngeal lesions have progressed considerably. Her nasal lesions have, however, remained stationary.

CASE 2.—J. H., a white unmarried man 53 years of age, was admitted to the hospital on Jan. 14, 1941, complaining of anosmia and hoarseness. He was born in Austria and at the age of 6 months was brought to this country by his parents, both of whom died of causes unknown. There were no siblings.

The patient's past history was interesting only in that he had had pneumonia in 1928, a tonsillectomy and adenoidectomy in 1933, a hemorrhoidectomy in 1940 and a "nose operation for obstruction" in 1940 (possibly a partial submucous resection). There was no history of scleroma in the family.

The present illness dated back to 1933, when the patient began to notice dyspnea on exertion and slight hoarseness. Anosmia began three years before his admission to the hospital, along with nasal obstruction. The obstruction was partially relieved by the "nose operation," but the anosmia persisted. Until about a year before his admission the dyspnea and hoarseness remained at a status quo, but shortly thereafter these symptoms became progressively worse. The patient was seen in the bronchoesophagologic clinic some ten months before admission, at which time the diagnosis of scleroma was made by biopsy of the affected nasal tissue. Biopsy of laryngeal tissue which appeared to be involved, and repeated biopsies since that time, failed to reveal the typical picture of scleroma. The patient was followed in the clinic until the time of admission, there being no improvement but rather a slow, steady progression of the disease in spite of fairly vigorous antisyphilitic therapy.

On admission the patient was found to be well developed and nourished, not acutely ill but suffering with severe hoarseness and slight dyspnea on exertion. Physical examination gave essentially negative results except for the nasal and laryngeal findings. The nasal septum was found to be markedly thickened. The inferior turbinates and lower portion of each side of the septum were covered with a grayish granulation type of tissue which was fairly soft and bled profusely when touched. There was considerable polypoid degeneration of both middle turbinates, and moderate crusting could be seen in both nasal fossae. A thin watery discharge emitting a peculiar insipid odor was present. The pharynx was not involved, but mirror examination revealed pronounced thickening of both false cords and moderate edema of both true cords, with considerable crusting and piling up of tissue in the posterior commissure. Subglottically, considerable crusting was present but no other pathologic changes could be seen.

The Wassermann and Kahn reactions were negative. Von Frisch bacilli grew profusely from nasal swabs. Urinalysis showed a trace of sugar, and the fasting blood sugar content was 125 mg. per hundred cubic centimeters; however, on a restricted carbohydrate diet, the urine remained sugar free and the blood sugar within normal limits. Other laboratory data were unimportant.

On Jan. 16, 1941 the patient was given 40 grains (2.6 Gm.) of sulfanilamide immediately and 15 grains (1 Gm.) every four hours. The next morning his blood concentration was only 4 mg. per hundred cubic centimeters and the dosage was increased to 30 grains (2 Gm.) every four hours. On January 20 the blood concentration had reached 12 mg. per hundred cubic centimeters, and the dosage was accordingly reduced to 25 grains (1.6 Gm.) every four hours. The blood level was then maintained at 8 mg. per hundred cubic centimeters for ten days, when a sudden drop in the hemoglobin and red cells accompanied by clinical icterus necessitated immediate discontinuance of administration of the drug. The icterus index at this time was 28 and the hemoglobin and red blood cells dropped from 100 per cent and 4,800,000 respectively to 70 per cent and 3,900,000. No other untoward sequelae were noted.

During the course of the drug therapy, considerable febrile reaction occurred. The temperature rose gradually over a period of seven days to 102 F., and then there was a gradual fall thereafter to normal. The pulse and respirations remained normal.

Subjectively the patient improved greatly. In addition, considerable regression of the nasal lesions occurred, along with marked lessening of thickening of the false and true cords. The hoarseness became much less, and the patient stated that he "hadn't felt so good in the past five years."

He was sent home for several weeks and then asked to return for more treatment. This he did on March 11, 1941. On admission this time the nasal and laryngeal picture was about the same as at the time of discharge except for the fact that the nasal granulomatous tissue had increased somewhat in extent. In addition, hoarseness had again become noticeable and subglottic crusting troublesome. Biopsies of the nasal scleromatous tissue revealed an apparent improvement, but the sections still showed scleroma to be unmistakably present. Again biopsy of laryngeal tissue did not show scleroma. Cultures of material from the nose yielded von Frisch bacilli.

The patient was given 60 grains (4 Gm.) of sulfathiazole immediately and 15 grains every four hours. The concentration in the blood the next morning was 4 mg. per hundred cubic centimeters, and this level was maintained for fourteen days. When no apparent improvement took place, 25 grains of sulfanilamide every four hours was substituted. The concentration in the blood the next morning was 10 mg. per hundred cubic centimeters, this level being maintained for eighteen days, when a decrease in hemoglobin and red and white blood cells, from 100 per cent, 5,100,000 and 5,000 respectively to 74 per cent, 3,400,000 and 3,800, necessitated discontinuing use of the drug.

As before during the course of sulfanilamide therapy, the temperature rose to a peak of 101 F. on the sixth day, with a gradual decline thereafter, while no febrile response occurred with sulfathiazole.

Subjectively, again the patient improved while receiving sulfanilamide but sulfathiazole apparently exerted no beneficial effect. Improvement in voice again occurred, as did regression of the nasal lesions. Von Frisch bacilli could be cultured throughout, but repeated biopsies of material from the nasal lesions were said to show "improvement."

CASE 3.—M. I., a 21 year old unmarried white man, born in Perth Amboy, N. J., of immigrant parents, was admitted to the hospital on April 1, 1941, complaining of progressive dyspnea and hoarseness of about three years' duration. His mother, a Pole by birth, died of pulmonary tuberculosis several years before, while his father, who died after a rib resection about five years before, was from

the Russian Ukraine. All seven siblings, one girl and six boys, were alive and well. No family history of scleroma could be elicited.

The patient had first presented himself at the bronchoesophagologic clinic eight months prior to his admission, and at this time the diagnosis of scleroma of the nose, pharynx and larynx was made by biopsy of tissue from the affected regions. Von Frisch bacilli were cultured also, their growth being profuse. His symptoms were first noted a little over a year before this visit to the clinic and consisted at first merely of crusting, foul discharge and nasal blockage. These symptoms became progressively worse, and in addition hoarseness appeared. A private physician was consulted and tonsillectomy advised and carried out. No relief was obtained, but rather his symptoms became worse until dyspnea became so pronounced as to necessitate emergency tracheotomy (Dec. 2, 1939; Manhattan Eye, Ear, and Throat Hospital), which gave immediate relief. After three months decannulation was performed, and though he remained very hoarse dyspnea was not extreme except after moderate exercise.

The patient was followed in the clinic for three months, and then it was felt that roentgen therapy was indicated. He was accordingly referred to the head and neck clinic of Dr. Hayes Martin at Memorial Hospital, where it was thought advisable to first carry out tracheotomy, since only a small glottic chink remained and glottic edema could be expected to follow roentgen irradiation. The patient refused to have a tracheotomy, and as substitute therapy he was given several injections of neoarsphenamine, without improvement. At our suggestion he returned to the Manhattan Eye, Ear, and Throat Hospital for the administration of sulfanilamide, since 2 patients suffering with scleroma had been so treated, with improvement.

On admission the patient was found to be moderately well developed and nourished but suffering with dyspnea of such severity as to cause marked respiratory retraction of the epigastrium and supraclavicular fossae. His nasal fossae were filled with foul crusts, the mucosa being atrophic but of fibrous consistency. The pharynx revealed typical scleromatous lesions, there being retracted scar tissue of almost cartilaginous firmness and grayish yellow hue covering the entire region and affecting to some extent the soft palate. The epiglottis, of infantile type, appeared to be drawn backward by scleromatous tissue in such a fashion as to cover the cords completely and thereby prevent indirect examination. However, by direct examination the cords were seen to be thickened, of dull reddish hue and practically immobile owing to arytenoid involvement. The glottis was reduced to an extremely narrow chink. In other respects the results of physical examination were essentially normal.

The Wassermann and Kahn reactions were negative. Other laboratory data were noncontributory.

The day after admission tracheotomy was carried out through the old scar. Specimens for biopsy were taken from the affected regions and reported as showing the picture of scleroma. Cultures were positive for von Frisch bacilli. Eighty-five grains (5.5 Gm.) of sulfanilamide was given immediately and 15 grains every four hours night and day thereafter. The next morning the level in the blood was 9 mg. per hundred cubic centimeters. This level was maintained for four days, and then it dropped to 6 mg. for an additional four days, at which time the dosage was decreased to half the previous amount. The concentration was then left at 4 mg. per hundred cubic centimeters for twelve days, when a steady drop in the hemoglobin and red blood cells, from 80 per cent and 4,000,000 respectively to 60 per cent and 2,900,000, necessitated discontinuing the administration of the drug.

Throughout the time the drug was being given the patient's temperature (rectal) fluctuated between 101 and 104 F., the highest temperature being noted during the first few days after administration of the drug was begun. His temperature on admission was 101 F., and after cessation of the sulfanilamide therapy it varied between 98.6 and 100 F. His pulse and respirations remained normal, and cyanosis was not troublesome.

Throughout the time the patient was receiving the drug he felt subjectively improved. In addition, his nasal crusting practically disappeared, the pharyngeal lesions appeared less pronounced and the vocal cords became less thickened, with increased mobility. Biopsy of material from the affected regions showed "improvement," while cultures showed a considerable lessening of the profusion of growth of von Frisch bacilli.

CASE 4.—A 56 year old man, an immigrant, was admitted to the hospital on April 25, 1941, with the complaint of progressive hoarseness of ten years' duration. He had been seen first in the clinic and repeatedly since 1935. Biopsies at intervals were said to reveal chronic inflammatory tissue. In 1939 a positive diagnosis of scleroma was made by biopsy, but the patient failed to return for therapy.

He responded to a letter sent to him during the past April and presented himself for admission. Sulfanilamide therapy was to be tried, but the patient, after taking the initial dose, refused further treatment and signed a release.

He was born in Italy and emigrated to this country in 1908. His father died of unknown cause, and his mother died from typhoid fever. Five brothers and two sisters were alive and well, there being no other siblings. His past history was not of interest except for the progressive hoarseness and dyspnea.

On admission the patient was found to be fairly well developed and nourished; he had moderate dyspnea and was extremely hoarse. The results of physical examination were essentially normal except for the nasopharyngeal and laryngeal findings. The nasopharynx was found to appear glazed, giving the typical picture of rather advanced scleroma. The cords were found to be markedly thickened, and motion was considerably impaired. In the posterior commissure there was considerable piling up of tissue, which on biopsy proved to be scleromatous.

Since the patient's discharge he has not been seen.

CASE 5.—A 62 year old Italian immigrant was seen in the clinic in April 1940. His complaint at that time was hoarseness. Examination of the larynx revealed thickening of the cords with heaping-up of tissue in the posterior commissure and pronounced crusting in that region. In addition, there was a contact ulcer on the left cord in the region of the vocal process.

Biopsy of a specimen taken from the heaped-up tissue of the posterior commissure showed scleroma. The patient has not returned to the clinic, however, in spite of repeated letters advising his return for therapy.

CASE 6.—G. C., a 34 year old white man, married, born in Brooklyn, who had never been out of this country, was admitted to the hospital on Feb. 2, 1942, complaining of hoarseness for the past seventeen years, difficult nasal breathing since childhood and dyspnea for the past six months.

Examination revealed extreme constriction of the nasal fossae due to swelling of the turbinates, which were covered with foul-smelling crusts, which were bluish, very hard and resistant. In the larynx the false cords were considerably swollen, which made it difficult to see the true cords. Direct examination showed that the cords were markedly thickened and dull red. There was a large bluish red mass subglottically on the posterior wall of the trachea. Biopsy of a specimen

taken from this mass as well as one from the lower turbinate showed scleroma. The patient was advised to enter the hospital for sulfanilamide therapy, but he was unable to do so at that time; however, he promised to come back as soon as he cleaned up his business affairs, which would take him two or three months.

CONCLUSIONS

1. The term rhinoscleroma should be changed to scleroma, as in most cases bearing this label there is involvement of the pharynx, larynx and treachea as well as the nose.

2. Scleroma is not geographically limited, as formerly thought, but may occur any place.

3. If the disease is kept in mind and material for biopsy taken, the diagnosis is not difficult.

4. Sulfanilamide and its derivatives seem to control the secondary infection but have no effect on scleromatous tissue.

ABSTRACT OF DISCUSSION

DR. FRANCIS CARTER WOOD, New York: There is very little that can be added to this excellent paper.

In this country the disease under consideration is usually recognized in the late stages, when the patient has extensive lesions and treatment is mostly hopeless except for moderate palliation. However, occasionally a cure is obtained in a case of long-standing scleroma. For example, a Russian woman whom I saw many years ago had her nasal passages treated with acid by an alleged nose and throat specialist for some years, with no improvement. The late Drs. Coakley and MacEnty made clinical diagnoses of carcinoma of the larynx. MacEnty did a biopsy, which showed rhinoscleroma involving the nasal passages, pharynx and larynx. She was referred to me for roentgen therapy. I administered over a period of four years some twenty treatments, giving the treatments very slowly and in small doses. MacEnty reported after the first year that the nose had practically healed but that there were small patches on the vocal cord. The irradiation was continued. A year later she said there was a small growth in the left nostril. He removed this, but microscopically there was no evidence of rhinoscleroma in it. It was a simple rhinolomatous area. The patient had no further trouble and died six years later, at the age of 69, of an intercurrent disease. That represents a favorable case, and I must confess that they are rare.

The difficult case is one of very extensive pharyngeal and laryngeal involvement in which the roentgen irradiation has been given too fast and in too large a quantity, with the production of a dense tissue sclerosis. In such a case a tracheotomy is frequently necessary, and the patient should be warned beforehand that that tracheotomy is apt to be permanent, because it is impossible or may be impossible for the fistula in the irradiated tissues to heal.

I have had a patient under observation for the last three years who is perfectly comfortable. The disease is under control, but nothing more. He still has a good deal of trouble. He obviously will have a permanent laryngeal fistula.

It is important, I think—and this paper is very timely in that respect—for otolaryngologists to know that there going to be a good deal more scleroma in this country than there has been in the past as cordial relationships are opened with

South America, because there are large numbers of patients afflicted with this disease in some of the Central and South American cities. So, there may be a drift to the north of a disease which is troublesome, almost always incurable and if curable only with the most prompt recognition, by means of biopsy. Apparently, clinically it is not characteristic enough for diagnosis.

Dr. F. W. Dixon, Akron, Ohio: Dr. Cunning has very thoroughly discussed the history and pathology of this chronic disease. It is interesting to note that sulfanilamide or a derivative does not stop its progress. Most otolaryngologists do not encounter so many cases as he has reported.

In the past fifty years the only form of treatment which has seemed to control the disease partially is radiotherapy. A review of the cases reported shows that some 24 patients have received this form of treatment; and of this number 21 have been improved, and 2 not improved and in 1 the result was doubtful. It is earnestly desired, however, that authors reporting cases of this disease state how long the improvement was observed. In many of the cases in which improvement was noted it was of less than three months' duration; in only 1 case was the period longer than three years.

The location of the lesions influences the duration of relief. The nose offers a better site for treatment than the trachea and bronchi. In 1 case I observed, in which the nose and pharynx were involved, irradiation controlled the progress for five years. In another, in which the nose and trachea were affected, roentgen therapy was given to the nose twice and to the trachea three times in eight years. However, the patients were comfortable during this time and the progress of the disease seemed controlled. In my cases these growths were removed surgically, after which radiation therapy was given. This seems to me better than irradiation alone, as the airways are not so obstructed by the reaction which follows this therapy.

Dr. Joseph I. Kemler, Baltimore: I had the good fortune of encountering a case of rhinoscleroma in this country, that of an American girl, about fifteen years ago, when I came here from Vienna. I have been very well posted on the disease.

This patient had been consulting physicians for several years, but the disease was unrecognized. As soon as I saw her I recognized that she had rhinoscleroma. I afterward proved it by pathologic examination. She had involvement of both nostrils, one being completely obstructed and the other only partially obstructed. The nasopharynx was filled up completely on one side, pulling up on the soft palate and uniting it with the rest of the tissue; the other side was still open, and she could breathe a little. She had epistaxis very frequently. That is the reason she consulted a physician. I sent her to a radiologist, who gave her radium. She has made a complete recovery, and to this day she has had no recurrence of her disease. The nose is practically normal except for some scar formation. The soft palate, of course, has more scar formations than the nose.

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FOREIGN BODIES OF EXTERNAL CANAL, MIDDLE EAR AND MASTOID AND THEIR COMPLICATIONS

REPORT OF THREE CASES

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Comprising an important percentage of every physician's practice are the complications resulting from accidental or avoidable trauma or infection. Forming no small part of this percentage are the accidents of children, who have a natural predilection for falling out of apple trees and stuffing foreign bodies into the ears, nose and throat. Frequently consulted in such instances is the otorhinolaryngologist. He is called on to remove stones, beads, beans, coins, weeds, pins and needles from a tiny air passage or esophagus. This too often necessitates the use of general anesthesia and direct or indirect laryngoscopy. There is the constant danger of injury to delicate membranes or of invasion by bacterial organisms. Numerous precise and delicate instruments have been perfected for this work.

Not so common in occurrence, but of equal importance in proper removal, are foreign bodies in the external auditory canal, which are sometimes pushed back into the middle ear or the mastoid region by clumsy efforts on the part of relatives or other unqualified persons. Here, especially, the resulting complications may be due not so much to the incident or the type of foreign body as to the method employed in its removal.

UNAVOIDABLE TRAUMA

Unavoidable trauma may be created by such objects as broken glass, sharp weeds, insects, tin, gravel and slag. The problem, then, is to remove the foreign body with the minimal degree of additional damage and to employ sufficient after-care to prevent bacterial infection. Tragic is the case of Chalier and Rousset,[1] who reported the death of a 3 year old child from tetanus after a small stone had been pushed into his external auditory canal. The stone was never removed.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., in 1942.

1. Chalier, J., and Rousset, J.: Foreign Body in the External Auditory Canal Causing Tetanus in a Child Three Years Old, Lyon méd. **141**:675 (June 10) 1928.

Unavoidable trauma in adults may be due to foreign bodies of a more ominous nature, such as knife blades, slag, bullets and shrapnel. The last is especially frequent in times of war.

AVOIDABLE TRAUMA

Persistent attempts at removal even of a smooth foreign body from the ear of a frightened, struggling child too often results in avoidable trauma. The walls of the canal become swollen, thereby holding the foreign body even more securely in place, and aggravation, due to continued manipulation, results.

Certain other foreign bodies, especially the organic ones, such as beans, when moistened by irrigation become many times their normal size. Rosenwasser [2] suggested repeated instillations of absolute alcohol to produce drying and shrinkage before removal is attempted.

Never forgotten will be my first experience with a wood tick at the very beginning of my medical career. The patient came in complaining of an earache. Examination disclosed the wood tick. The diagnosis was simple. So, too, should have been the treatment. I reached in with a forceps and pulled. Out came the wood tick, but it brought with it the greater portion of the tympanic membrane!

Insects, especially wood ticks, which are encountered frequently in certain localities, should be killed first by instilling a drop or two of chloroform into the external auditory canal. One drop is usually sufficient to kill the insect and still not blister the drum. The dead invader may then be lifted out with forceps or removed by gentle irrigation, thus eliminating the hazard of injury to the tympanic membrane. Richards [3] stated that he used a mixture of chloroform and oil.

Hilton [4] reported an instance in which an eraser from the end of a lead pencil was incompletely removed from the external auditory canal of a young boy. One year later the ear started to discharge and continued to do so for three years. He had attacks of severe pain and at times was irrational. A radical mastoidectomy was performed. Wedged into the annulus tympanicus was a large portion of the eraser. The dura was exposed in the middle ear, which was filled with polyps and granulations. All of these were removed. Convalescence, apparently, was uneventful.

A similar case was encountered in my own practice. I saw the patient after some one else had attempted removal of the eraser, with

2. Rosenwasser, H.: Foreign Bodies in the External Auditory Canal, Am. J. Surg. **36**:96 (April) 1937.

3. Richards, G. L.: Foreign Bodies in the External Auditory Canal, Am. J. Surg. **6**:514 (April) 1929.

4. Hilton, G. E.: A Foreign Body in the External Auditory Canal, Canad. M. A. J. **33**:308 (Sept.) 1935.

the use of general anesthesia. Pieces had been broken off and pushed through the drum. The patient was subjected to another general anesthesia and the pieces of foreign body were removed with alligator forceps and a curet. Otitis was present for two or three weeks but cleared up and has not returned. The drum, however, remained perforated, and the child was left with a 25 per cent loss of hearing.

REVIEW OF THE LITERATURE

The English literature contains 2 interesting cases of foreign body in the mastoid. A case of a pussy willow catkin in the ear of a 5 year old boy was reported in 1938. [5] The first physician to treat the patient aurally removed only part of the foreign body. Some time later more was removed with use of general anesthesia, on which occasion perforation of the drum was noted. After a fortnight the ear commenced to discharge and was treated with boric acid and iodine for about six months. Then a simple mastoidectomy was performed. The remainder of the pussy willow catkin was found lodged in the aditus and was recovered and removed. Although the otitis improved, the ear drum remained perforated.

In the second case, reported by Faulder,[6] both a simple and a radical mastoidectomy were required for complete removal of a bead from the antrum of an 8 year old boy. Just how the bead came to be there was never satisfactorily explained.

Longaker [7] reported a similar case in the American literature. A boy of 6 placed a glass button in his ear. Attempts to remove it were unsuccessful. Roentgenograms revealed no foreign body, and surgical intervention was not attempted. The ear continued to suppurate during the next five years and repeated myringotomies were performed. Prior to being seen by Dr. Longaker, the boy was pushed against a clothes hanger. The injury sustained at that time resulted in an exacerbation of his symptoms, and a modified radical mastoidectomy was performed. During the course of the operation, a pea-sized ball of cotton and a split pea–sized piece of button were removed. Convalescence was uneventful except for considerable impairment in hearing.

Also in the American literature is the report of Hempstead [8] on 3 cases of bullets in the ear. In the first instance the foreign body caused

5. Bettington, R. H.: Peculiar Sequel to a Foreign Body in the Ear, M. J. Australia **1**:535 (March 19) 1938.

6. Faulder, T. J.: Foreign Body in Mastoid Antrum, Proc. Roy. Soc. Med. **14**:26, (Nov.) 1920.

7. Longaker, E. P.: Two Interesting Foreign Body Cases of the Mastoid and Nose, Ann. Otol., Rhin. & Laryng. **38**:1158 (Dec.) 1929.

8. Hempstead, B. E.: Bullets in the Ear: Report of Three Cases, J. A. M. A. **98**:2281 (June 25) 1932.

a discharge almost immediately. A constant source of trouble, nevertheless its removal did not take place until sixteen years after the actual injury. In the second case the middle ear tolerated the bullet for five years before a discharge was apparent. In spite of an end to end anastomosis done to correct the resulting facial paralysis three months after the shooting, the bullet was not removed until five years later. At that time a radical mastoidectomy had to be performed. In the third instance eight years elapsed before the bullet caused sufficient trouble to warrant radical mastoidectomy for its removal.

Martin,[9] in his paper on surgical repair of the facial nerve, described the presence of lead in the mastoid as the result of bullet injuries. These foreign bodies apparently caused no symptoms which in themselves warranted surgical intervention. Other men,[10] in performing surgical repair of the facial nerve, have had similar experiences.

The case of a living worm removed from the mastoid of an 8 year old girl is reported in the German literature.[11] There had been no previous indication of the presence of a foreign body. Surgical treatment for acute mastoiditis following scarlet fever had been indicated. The worm was encountered during the surgical procedure and was removed in two lengths. Each segment measured approximately 2 cm. in length and up to 2 mm. in width.

FOREIGN BODY OF THE MIDDLE EAR AS AN INDUSTRIAL HAZARD

Unemphasized in the medical industrial literature but becoming an increasingly important industrial hazard among men who do acetylene welding is the entrance of slag into the mastoid region.

Rich in detail and comprehensive in scope is the article on electric and gas welding by Britton and Walsh.[12] Of principal importance to them, however, were the effects of welding on the eyes and lungs, for which proper protective measures are being taken. Casually mentioned was the occurrence of otitis media following perforation of the ear drum by molten metal. On the basis of my own experience, I feel that this danger is rapidly becoming more important with the development of this industry.

Loss of hearing has serious psychologic and economic reverberations on the persons affected. Partial or complete deafness may be a

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9. Martin, R. C.: Recent Experiences with Operation on the Facial Nerve, Arch. Otolaryng. **32**:1071 (Dec.) 1940.

10. Viole, P.: Personal communication to the author.

11. Steinmann, K. B.: A Worm in the Mastoid, Monatschr. f. Ohrenh. **65**: 222, 1931.

12. Britton, J. A., and Walsh, E. L.: Health Hazards of Electric and Gas Welding, J. Indust. Hyg. **22**:125 (April) 1940.

serious aftermath of burning of the tympanic membrane by slag. This has been pointed out in reports by Lion [13] and Karashima.[14]

Heermann [15] expressed the belief that the injury may be more prevalent than is suggested by medical reports, inasmuch as the majority of patients so affected consult a physician only after secondary infection has occurred. His report of 15 cases includes those of blacksmiths and foundry workers doing iron casting, as well as of welders.

My observations are based on the treatment of 13 welders suffering from burns of the tympanic membrane due to slag. The majority of these patients suffered injury to the left ear; the right ear was affected in left-handed workers.

The minute pieces of hot metal and vitrified cinders fall into the upturned auditory canal, burn their way through the tympanic membrane and lodge in the mastoid bone. The resultant perforations vary from an area the size of a pinpoint to the entire central portion of the drum.

Intense pain, swelling and redness of the tympanic membrane are the first symptoms. About three days later discharge occurs. It is usually profuse, and washed out by it are the tiny foreign bodies responsible for the damage. After twenty-four hours the discharge may cease and the ear remain dry. The average length of time required for recovery is three weeks. However, the slag may remain buried in the mastoid. Discharge will then continue spasmodically until all of the foreign body has been expelled. One of my patients had a profuse discharge for fifteen months.

Next to prevention in importance, immediate proper treatment is necessary to preserve normal hearing. In the 13 patients I treated, all but 4 of the perforations closed. These 4 did not heal, although the ears remained dry. The fault may lie in the fact that the 4 men affected remained at home under the care of the family physician before consulting an otologist. About ten days elapsed in each case. In 1 patient a false membrane formed but subsequently opened three times before healing because of recurrent attacks of discharge. Only 1 of the 4 men was left with residual hearing of 20 decibels.

Even in cases of severe injury the associated temperature has not been known to rise above 99.5 F. Treatment consists merely of gentle cleansing of the auditory canal once or twice a week. Any form of applied medication or irrigation of the ear proves irritating and prolongs

13. Lion, H.: Burn of the Tympanic Membrane in Welder, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:195 (Aug.) 1929.

14. Karashima, N., cited by Heermann.[15]

15. Heermann, J.: Cases of Burns of the Auditory Canal, Middle Ear and Labyrinth Occurring in Welders and Foundry Workers, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:546 (July) 1936.

the ailment. Tincture of mercresin [16] was used in 1 or 2 instances of secondary infection of the auditory canal.

In questionable cases the presence or absence of slag in the mastoid may be demonstrated by roentgenograms. Laboratory findings are without significance.

With the exception of the 4 men treated by their family physicians, none of the welders was required to remain away from work. Cotton placed in the external auditory canal served as a precautionary measure against further hazard from falling slag. This form of prevention, or one similar to it, should be routine and compulsory, just as the wearing of masks and dark glasses to protect the eyes from the intense light of acetylene and electric welding has become a requirement.

REPORT OF CASES

CASE 1.—N. G., a Mexican aged 38, was admitted to the County Hospital on June 19, 1933, after a street fight, in which he was an active participant. Emergency examination revealed the presence of a deep knife wound anterior to the left ear and penetrating back through the external canal. There was profuse hemorrhage.

At the preliminary examination the patient's temperature and reflexes were normal. The canal was packed and the lips of the wound sutured. He was given tetanus antitoxin and put to bed.

Four days later the picture had changed. The patient's temperature was 103.6 F., the pulse rate 102 and the respiratory rate 20. He was transferred to the ear, nose and throat department, where he came under my attention. At that time he was in a semiconscious condition, with definite rigidity of the neck. The Babinski and Kernig signs were present.

Spinal puncture demonstrated increased pressure of the spinal fluid, with a cell count of 18,600, of which 95 per cent were polymorphonuclear leukocytes. There were a decrease in sugar and an increase in globulin, but no bacteria.

Roentgenograms revealed the presence of a foreign body below the auditory meatus, in the region of the middle fossa.

A simple mastoidectomy was performed immediately, without the administration of an anesthetic. The tip of a pocketknife blade was found just above the jugular bulb in the middle fossa, in close proximity to the facial ridge. Because of the extremity of the situation and the patient's moribund condition, the facial ridge was sacrificed to facilitate removal of the foreign body. The area was well exposed and the incision left open for drainage. Almost immediate improvement was shown, and by the end of the first week the temperature remained within normal limits.

However, although the posterior incision healed, the middle ear continued to discharge. A radical mastoidectomy was therefore performed on July 26 and all infected tissues were removed. The wound was closed with the usual skin graft. Convalescence was uneventful except for paralysis of the left side of the face for which the patient refused further surgical treatment.

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16. Mercresin has the following formula: secondary amyltricresols, 0.1 per cent; orthohydroxyphenylmercuric chloride, 0.1; acetone, 10 per cent, and alcohol, 60 per cent.

CASE 2.—J. R., a Mexican aged 60, entered the County Hospital on Sept. 2, 1930. He gave a history of having been shot behind the left ear thirteen years before. Apparently, during the time elapsed his ear gave him little trouble. One week before coming into the hospital there suddenly developed headache, considerable swelling around the left ear, with associated purulent aural discharge, and elevation of temperature.

Examination revealed that the left auditory canal was filled with purulent discharge. There was also pronounced postauricular swelling.

Roentgenograms revealed a large number of foregn bodies scattered throughout the mastoid area. There was also definite evidence of destruction of the mastoid cells.

Routine examination of the blood revealed a white cell count of 20,000 per cubic millimeter and a 4 plus Wassermann reaction.

Antisyphilitic treatments were instituted, and a simple mastoidectomy was performed. Fifteen fragments of lead bullet were removed from the mastoid region. An epidural abscess was encountered during the surgical procedure and was opened and drained. The postauricular wound was closed. It healed in three weeks.

Although the patient's temperature dropped and the swelling improved, the middle ear continued to discharge. A radical mastoidectomy was performed on December 10. The cavity was thoroughly cleansed and the usual skin graft made. Recovery was uneventful.

CASE 3.—L. M., a white man aged 45, while welding on Feb. 26, 1941, had a spark fly into his left ear. Examination a few hours later revealed slight abrasion of the external auditory canal on the left side. A small piece of steel shot was visible in the left tympanic cavity through a moderate-sized perforation in the anterior margin of the drum.

On the following morning a curved probe was introduced into the canal and directed toward the foreign body. Then the current of a giant electric magnet was turned on. By this method the steel fragment became magnetized and was easily removed. It measured from 5 to 8 mm. in diameter.

Immediate impairment of hearing was noticed at the time the steel shot entered the ear. After removal of the foreign body the patient could hear the whispered voice on contact.

On March 3 a moderate amount of purulent discharge appeared from the left middle ear. On March 6 there was increased destruction of the left tympanic membrane. By March 8 only a small rim of drum remained attached to the annulus tympanicus at the posterior half. The discharge has steadily decreased.

The patient has been under conservative treatment, with no irrigations. After cleansing the ear, iodine or sulfanilamide powder was insufflated.

COMMENT

Too much emphasis cannot be placed on the importance of careful removal of foreign bodies from the external auditory canal. This is particularly true when one is dealing with frightened children.

Forceful methods aggravate the traumatic conditions and increase the patient's apprehension. The better method and the better results are obtained by the simplest procedure. A radical mastoidectomy should

not become necessary in order to remove a foreign body that has entered by way of the external auditory canal.

Some of the avoidable and unavoidable causes of trauma to the tympanic membrane, middle ear and mastoid region have already been pointed out. One other deserves mention. Too many otologists, and this is true of practitioners in other branches of medicine, are prone to invent their own pet instruments either for surgical procedures or for the removal of foreign bodies. The perfection and invention of new surgical tools are commendable, but due conservatism should be shown. Frequently more damage is done with that special ear spoon of the otologist than is caused by the foreign body so snugly lodged in the canal or the middle ear. This may be particularly true of that elusive piece of cotton patients with chronically discharging ears are always putting in the auditory canal and forgetting to remove.

A considerable portion of this paper has been devoted to the presence of burns of the tympanic membrane due to the entrance of slag into the external auditory canal of welders. This industrial hazard should receive the proper attention of medical men, particularly otologists, until the necessary protective measures become a part of the industry. I believe this can be accomplished by reporting all such cases in the literature, where they can be brought to the attention of the proper authorities.

The 3 case histories are presented as illustrative of foreign bodies in the mastoid and the middle ear and are self explanatory.

34 North Ash Street.

TREATMENT OF CYSTS OF THE LARYNX

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In December 1939, Erich and I [1] reported on the treatment of a congenital laryngeal cyst in the case of a girl 17 years of age. The cyst was removed by means of lateral thyrotomy. Since that time, 2 additional cases of this type have been encountered at the Mayo Clinic.

Laryngeal cysts form 4.9 per cent of all laryngeal tumors. There are three types of laryngeal cysts, namely, mucous, hemorrhagic and congenital. Mucous cysts are caused by inflammatory obstruction of the ductal portion of a mucous gland. They are encountered most frequently in the vicinity of the epiglottis, where the mucous membrane contains abundant mucous glands.

The treatment of the mucous cyst and the hemorrhagic cyst is a matter of removing the tumor under suspension laryngoscopy. Surgical diathermy may be used if necessary. The treatment of the congenital laryngeal cyst depends on the size and extent. The Lynch suspension apparatus may be employed in the care of the smaller ones. The large cysts are better removed by means of lateral thyrotomy, as is indicated in the following report.

REPORT OF TWO CASES

CASE 1.—A man aged 58, on admission to the Mayo Clinic, gave a history of progressive hoarseness for three years. For the past six months he had had difficulty in breathing on exertion. Examination revealed a cystic mass on the left side of the larynx, which completely filled the glottis and extended above it. There was a cystic tumor in the left piriform sinus (figs. 1 and 2). A tomograph revealed that the tumor filled the supraglottic region and extended from just above the vocal cords to the hyoid bone (fig. 3). Preliminary tracheotomy was performed, and a cannula was inserted on account of the obstruction. Ten days later, a midline incision was made and the thyroid cartilage was divided at the junction of the anterior and the middle third. The saw ruptured the cyst after passing through the thyroid cartilage, and the thick mucus-like material was evacuated (fig. 4). The wall of the cyst was dissected by means of a cotton applicator and entirely removed without entering the laryngeal mucosa. A Penrose drain was inserted into the bed of the sac, and the soft tissues over the thyroid cartilage were closed with fine catgut. A split rubber tube was inserted to prevent surgical

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From the Section on Laryngology, Oral and Plastic Surgery, the Mayo Clinic.

Presented by means of moving pictures at the Forty-Eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., June 2, 1942.

1. New, G. B., and Erich, J. B.: Congenital Cysts of the Larynx: Report of a Case, Arch. Otolaryng. **30**:943-949 (Dec.) 1939.

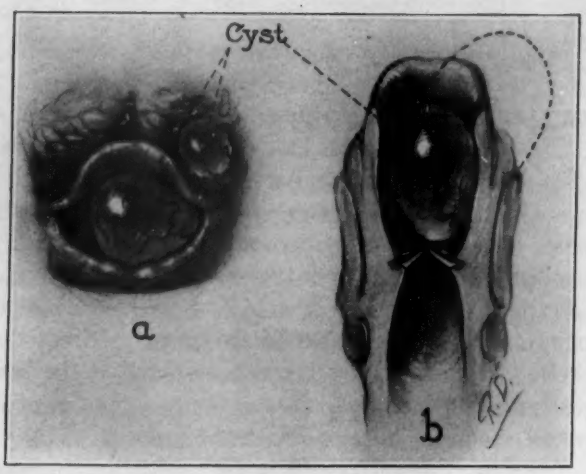

Fig. 1 (case 1).—Extent of the cyst.

Fig. 2 (case 1).—Cyst of the left side of the larynx. Photograph made under laryngeal suspension.

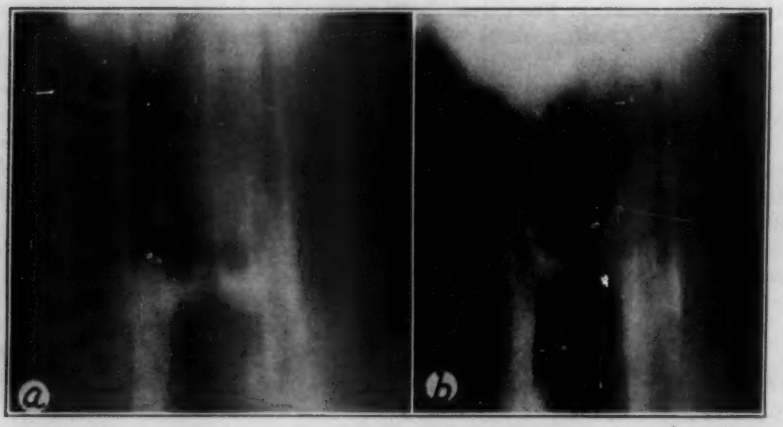

Fig. 3 (case 1).—Tomographs: (*a*) congenital cyst on the left side of the larynx; (*b*) larynx six months after operation, with normal contour.

Fig. 4 (case 1).—A midline incision was made, and the thyroid cartilage was divided at the junction of the anterior and the middle third. An electrically driven circular saw is shown cutting the left thyroid cartilage for exposure of the cyst. The index finger points to the notch of the thyroid cartilage.

Fig. 5 (case 2).—Cyst and enlargement of the right arytenoid region. Photograph made under laryngeal suspension.

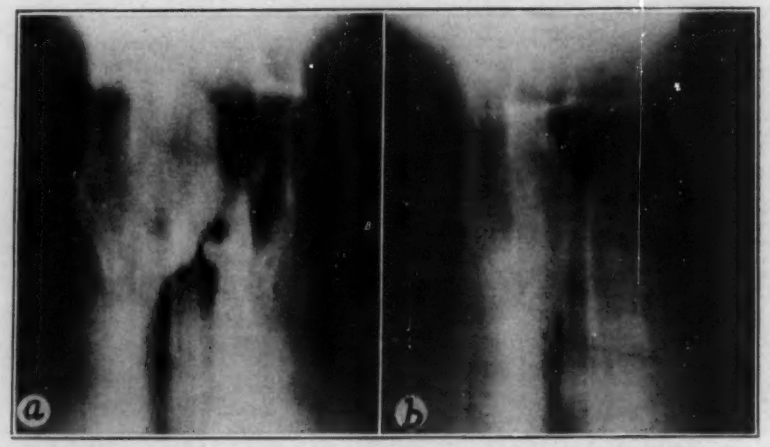

Fig. 6 (case 2).—Tomographs: (*a*) cystic mass present before operation; (*b*) appearance of larynx three weeks after operation.

emphysema. The skin was closed with silk, and the tracheal tube was replaced. The patient made an uneventful recovery. A tomograph of the larynx made one month later revealed slight fulness on the left lateral wall. In the tomograph made six months later the contour of the larynx was normal (fig. 3). On examination at that time the larynx was normal and the patient had a good, clear voice.

CASE 2.—A woman aged 40 gave a history of hoarseness for two and a half months. Before she came for examination, her condition had been diagnosed as a malignant tumor of the larynx, and ten roentgen treatments had been given. The treatments had produced considerable actinodermatitis.

Examination at the clinic revealed a cystic mass on the right side of the larynx, which filled the glottis and caused bulging of the right arytenoid region (figs. 5 and 6 *a*). The tumor was removed in the same manner as described in case 1, after lateral thyrotomy. The patient made an uneventful recovery. A tomograph was made three weeks after operation (fig. 6 *b*). Although the right side of the larynx was still thickened slightly, the patient had an excellent voice after the operation.

Lateral thyrotomy is a satisfactory approach for removal of congenital laryngeal cysts.

EFFECTS OF VARIOUS TYPES OF MOTION ON DIFFERENCES IN HYDROSTATIC PRESSURE BETWEEN ENDS OF A SEMICIRCULAR CANAL

A THEORETIC ANALYSIS

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In this paper a semicircular canal has been idealized, by making the following assumptions: (1) There is no flow of endolymph in the canal. (2) Response to motion is due to the pressure changes produced at the ends of the canal. (3) The walls of the canal are rigid. (4) The endolymph is incompressible and fills the entire canal. The mathematical analysis for every possible type of motion shows that pressure differences between the ends of the canal are produced only by linear acceleration or by angular acceleration or velocity about axes oriented in specific ways in respect to the canal. The pressure differences thus arising in the canal may be presumed to be a source of nerve stimuli. Substitution of experimental results in the derived equations should provide a test of the practical validity of the assumptions. Thus evidence can be obtained to determine whether or not there is an endolymphatic flow in the production of nerve stimuli in the semicircular canals.

Present knowledge of the physiology of the vestibular labyrinth is not adequate for a complete explanation of the operation of this apparatus. In the case of the semicircular canals it is well established that they respond to angular acceleration; however it is not certain to what extent angular velocity and linear acceleration produce stimuli. Furthermore, the manner in which the receptors in the cristae of the canals are stimulated is not definitely known. It is reported from tests and in the literature that there is an actual flow of the endolymph in the canals. On the other hand, glass models of the canals have failed to show a flow of the contained liquid [1]; furthermore, experiments by Ewald and Loeb have

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1. Maier, M., and Lion, H.: Arch. f. d. ges. Physiol. **187**:47, 1921.

demonstrated that normal responses in the canals are not prevented by section, ligation or plugging.[2] It would therefore seem that nerve stimuli result from compressional waves set up in the endolymph. In other words, response results from the pressure changes that may be produced by certain types of motion.

In this paper we are interested in treating mathematically the effects in the semicircular canals of various types of motion. It will be necessary to make certain assumptions in regard to the structure of the canals. It will be assumed that there is no flow of the endolymph in the canals and that response to motion is due to pressure changes produced at the ends of the canals. The possibility of a similar effect being produced in the utricle will not be considered at present even though it is thought by some that such may be the case. For the mathematical treatment of the pressure effects a canal will be represented as in figure 1, i. e., as a semicircular tube lying entirely in one plane with a pressure-measuring device

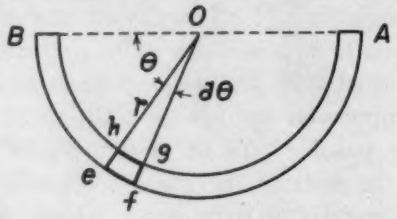

Figure 1

at the end. The walls of the canals will be assumed to be rigid and the endolymph incompressible and filling the entire canal. If there is no flow of the endolymph, its viscosity does not enter into the problem.

First, let us find the difference in pressure at A and B due to a constant counterclockwise angular acceleration, α, of the canal about an axis perpendicular to the plane of the canal through O, the center.

α is represented by a vector perpendicular to the plane of the canal and pointing out of the paper. Let S, which equals $\frac{\pi}{4}\overline{eh}$,[2] be the area of cross section of a canal, r the radius and ρ the density of the endolymph. Now consider the force due to the acceleration, α, on an infinitesimal element, $efgh$, of mass dm and of length $ef = dl$, which subtends an angle, $d\theta$, at the center of the circle. The mass of the element may be expressed

2. Ewald, J. R.: Physiologische Untersuchungen über das Endorgan des Nervus octavus, Wiesbaden, J. F. Bergmann, 1892. Loeb, J.: Arch. f. d. ges. Physiol. **49**: 175, 1891. For an excellent discussion and bibliography see: Hyndman, O. R.: Physiology of the Vestibular Labyrinth, Arch. Otolaryng. **29**:759 (May) 1939.

as follows: $dm = \rho S dl = \rho S r d\theta$. As the canal rotates, the wall of the canal constrains the element dm to move in a circle by exerting on it a centripetal force, $-dF_N$, toward the center, and the element dm exerts a centrifugal force, $+dF_N$, away from the center, on the wall of the canal. The subscript N is used to indicate that the force is perpendicular to the canal, and the negative and positive signs, to indicate whether or not it is toward or away from the center. The forces $-dF_N$ and $+dF_N$ depend on the angular velocity only and may thus be present even when there is no angular acceleration. These forces contribute nothing to the difference in pressure between A and B.

To give the element dm acceleration along its circular path, a tangential force, $-dF_T$, must act on the element. Conversely, the element must exert a force of reaction, $+dF_T$, equal in magnitude but opposite in direction to $-dF_T$ on whatever is pushing the element in a tangential direction—which in this case is the adjoining endolymph. The subscript T is used to indicate that the force is parallel or tangent to the canal. Forces directed toward A are taken as negative and forces directed toward B as positive. The force dF_T will be transmitted through the fluid to the end B. Accordingly, the force exerted at B due to tangential force will be

$$dF_T = r\alpha dm.$$

Putting in the aforementioned value of dm, we obtain the equation

$$dF_T = \rho S r^2 \alpha d\theta.$$

The pressure transmitted is

$$dp = \frac{dF_T}{S} = \rho r^2 \alpha d\theta.$$

The total pressure due to all the endolymph in the canal is thus

$$p = \int dp = \int_0^{\pi} \rho r^2 \alpha d\theta = \rho r^2 \alpha \pi = 2\rho \alpha A_1 \qquad (1)$$

in which A is the area enclosed by the canal and the diameter of the circle.

It is apparent that under ordinary conditions a canal is not likely to be subjected to an angular acceleration about its center as in the case just treated. Other axes of rotation will therefore be considered.

Next let the canal rotate about a fixed axis through O' (fig. 2) perpendicular to the plane of the canal. O' is so chosen that OO' is a perpendicular bisector of AB. Let d equal OO' and let α be the angular acceleration in a counterclockwise direction of the canal about O'. The radius of rotation of any element $efgh$ is now R. Let the mass of the element be dm and its length $dl = rd\theta$. The path traveled by the element dm is indicated by the dotted line, DE.

The force of reaction exerted by dm tangent to this path is

$$df_T = aRdm = aR\rho Srd\theta.$$

The force perpendicular to this path is

$$df_N = R\omega^2 dm = R\omega^2 \rho Srd\theta \qquad (2)$$

in which ω is the angular velocity about O'. If the canal starts from rest and if a is uniform ω equals at. In order to find an expression for the pressure increase at B it is necessary to consider the force dF_T tangent to the canal. This may be obtained by taking the components of df_N and df_T along the canal. Let ϕ be the angle which df_N makes with the normal to the canal at the point e. The component of df_N in the direction of dF_T is therefore $-df_N \sin \phi$. Since at the symmetric point on the other side of the canal the component is $+df_N \sin \phi$, it becomes evident that in

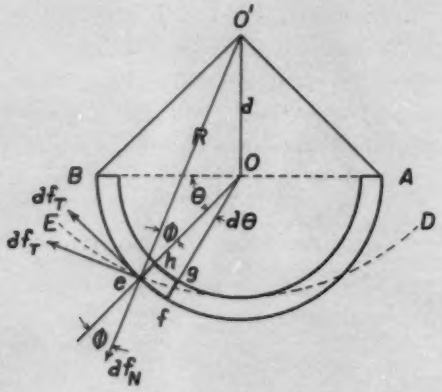

Figure 2

considering the canal as a whole, the components of df_N along dF_T on one side are neutralized by the components of df_N on the other side of the canal. So the normal force df_N contributes nothing to the pressure effect at B.

The component of df_T along dF_T is $df_T \cos \phi$. So the total pressure effect at B is

$$p = \frac{2}{S}\int_0^{\frac{\pi}{2}} df_T \cos \phi = \frac{2}{S}\int_0^{\frac{\pi}{2}} Ra\rho Sr \cos \phi d\theta.$$

In order to carry out the integration it is necessary to express $\cos \phi$ in terms of θ. By the theorem of cosines

$$d^2 = r^2 + R^2 - 2rR \cos \phi$$

and

$$R^2 = r^2 + d^2 - 2rd \cos (90 + \theta).$$

Adding these equations and solving for $\cos \phi$, we have the equation

$$\cos \phi = \frac{r + d \sin \theta}{R}.$$

Substituting this value of $\cos\phi$ in the foregoing integral, we obtain the equation

$$p = 2a\rho r\int_0^{\frac{\pi}{2}} (r + d\sin\theta)\, d\theta = a\rho\pi r^2 + 2a\rho rd = 2\rho aA_2 \qquad (3)$$

in which A_2 is the area enclosed by the canal and the lines $O'A$ and $O'B$.

We shall now treat the more general case as shown in figure 3, in which the axis of rotation through O'' is perpendicular to the plane of the canal as in the previous cases, but OO'' is not perpendicular to AB. Let b equal $O'O''$ and let d' equal OO'', which equals $\sqrt{b^2 + d^2}$.

If the canal be rotated about O through an angle $\mathrm{B} = \arctan\frac{b}{d}$, it will be seen that this case becomes similar to that of figure 2. Consider

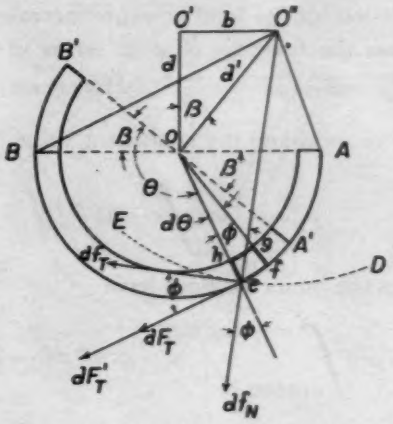

Figure 3

the element $efgh$ of mass dm and length $ef = dl$. The path traveled by the element is indicated by the dotted line DE, and the radius of rotation is R. The force of reaction exerted by the element tangent to the path is

$$df_T = aRdm = aR\rho Srd\theta.$$

θ is now measured from $B'A'$ instead of BA. The force perpendicular to the path traveled by the element is

$$df_N = R\omega^2 dm = R\omega^2\rho Srd\theta \qquad (2)$$

in which ω is the angular velocity about O''.

The integral of dF_T, the component of df_T tangent to the canal, is the same as in the case of figure 2 except that the limits of integration are different. The integral now becomes

$$p_T = \int dF_T = \tfrac{1}{S}\int_{\mathrm{B}}^{\pi+\mathrm{B}} df_T\cos\phi.$$

Putting in the equivalent value of df_T and expressing $\cos \phi$ in terms of θ, we obtain the equation

$$p_T = a\rho r \int_{\arctan \frac{b}{d}}^{\pi + \arctan \frac{b}{d}} (r + d' \sin \theta)\, d\theta.$$

Hence,

$$p_T = a\rho\pi r^2 + 2a\rho rd. \tag{4}$$

Let dF'_T be the component of df_N tangent to the canal. Hence $dF'_T = df_N \sin \phi$.

It is now necessary to find the pressure contributed at B by the integral $p_N = \frac{1}{S}\int dF'_T = \frac{1}{S}\int df_N \sin \phi$ and add it to equation 4 in order to obtain the expression for the total pressure increase at B. Again it is necessary to express the function of ϕ in terms of θ. By use of the theorem of sines, $\frac{\sin \theta}{d'} = \frac{\sin (90+\theta)}{R} = \frac{\cos \theta}{R}$. Hence $\sin \phi = \frac{d' \cos \theta}{R}$. Substituting this value of $\sin \phi$ and the equivalent value of df_N, we have the equation for p_N:

$$p_N = \omega^2 \rho r d' \int \cos \theta d\theta.$$

Integrating between the limits for θ we have

$$p_N = \omega^2 \rho r d' \int_{\arctan \frac{b}{d}}^{\pi + \arctan \frac{b}{d}} \cos \theta\, d\theta = 2\rho br\omega^2. \tag{5}$$

The value for the total pressure increase at B thus becomes

$$\left.\begin{aligned} p &= p_T + p_N = \pi a\rho r^2 + 2a\rho rd + 2b\rho r\omega^2 \\ \text{or} \qquad p &= 2a\rho A_3 + 2\rho br\omega^2 \end{aligned}\right\} \tag{6}$$

in which $A_3 = \pi r^2 + rd$ the area enclosed by the canal and sides $O''A$ and $O''B$.

A generalization may now be made for obtaining the value of the pressure increase at B for counterclockwise angular acceleration of the canal about any axis perpendicular to its plane and in any quadrant. If the center, O, of the canal is taken as the origin of coordinates, the x axis along AB and the y axis along OO', the value of the pressure increase, p, at B is obtained by substituting in equation 6 the coordinate values, b and d, for the location of the axis.

In the foregoing cases it has been assumed that a canal is a semicircle. Since the canals deviate somewhat from this simple curve, we

shall now consider an irregular canal as shown in figure 4 in order to determine the error resulting from assuming the circular shape. Let the axis of rotation intersect the plane of the canal perpendicularly at any point O''. Let α be the angular acceleration of the canal in a counter-clockwise direction and ω the instantaneous angular velocity. The centripetal force on any element $efgh$ of mass dm and length $dl = ef$ is

$$df_N = R\omega^2 dm = R\omega^2 \rho S dl$$

in which R is the radius of rotation of the element. The force tangent to the dotted line DE, or the circular path of the element about the axis through O'', is

$$df_T = \alpha R dm = \alpha R \rho S dl.$$

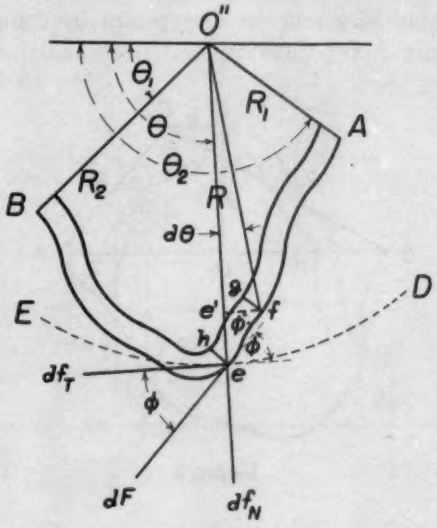

Figure 4

Let ϕ be the angle between the tangent to the circular path of the element and the tangent to the canal at the point e. If dF is the total force tangent to the canal at the point, then

$$dF = df_T \cos\phi + df_N \sin\phi.$$

From figure 4 it may be seen that $\cos\phi = \frac{e'f}{ef} = \frac{Rd\theta}{dl}$ and $\sin\phi = \frac{e'c}{ef} = \frac{dr}{dl}$. Substituting the values for the functions of ϕ and for df_N and df_N in the expression for dF, we obtain the following equation:

$$dF = \rho S \alpha R^2 d\theta + \rho S R \omega^2 dR$$

or

$$dp = \frac{1}{S} dF = \rho \alpha R^2 d\theta + \rho R \omega^2 dR.$$

Integrating between the proper limits, we obtain the value for the pressure increase at B:

$$p = \rho a \int_{\theta_1}^{\theta_2} R^2 d\theta + \rho\omega^2 \int_{R_1}^{R_2} R dR.$$

Since the definite integral $\int_{\theta_1}^{\theta_2} R^2 d\theta$ is twice the area, A_4, enclosed by the canal and R_1 and R_2, the expression for p finally becomes with the completion of the integration of the equation

$$p = 2a\rho A_4 + \tfrac{1}{2}\rho\omega^2 (R_2^2 - R_1^2). \qquad (7)$$

The magnitude of the error resulting from the assumption that a canal is semicircular may now be determined by comparing equations 6 and 7. In figure 5 are superimposed two canals, one of the shape

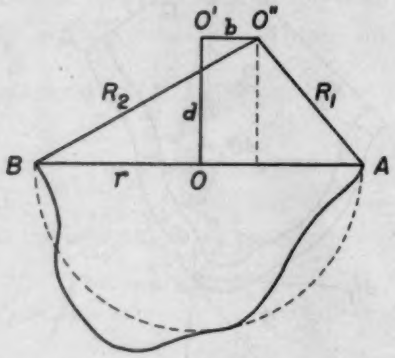

Figure 5

shown in figure 4 and one semicircular as represented by the dotted half circle. From this diagram it is seen that $R_2^2 = (r + b)^2 + d^2$ and $R_1^2 = (r - b)^2 + d^2$. Subtracting these equations,

$$R_2^2 - R_1^2 = 4rb.$$

By substitution, the term $\frac{1}{2}\rho\omega^2 (R_2^2 - R_1^2)$ in equation 7 becomes $2\rho br\omega^2$, and this is the same as the second term of equation 6. The difference between equations 6 and 7 is therefore in the quantities A_3 and A_4. A little consideration will show that the only difference between these two quantities is the difference of the areas enclosed by the canals and the line BA. Since a canal does not depart greatly from a semicircle, this difference is small and the error introduced by assuming the semicircular shape is therefore small.

In all the cases considered thus far the vector representing the angular acceleration has been perpendicular to the plane of the canal or, in other words, the axis of rotation has been perpendicular to the plane of the canal. These are special cases, and rotation about other axes must be considered. Take the case in which the axis is parallel to and lying in the plane of the canal—figure 6. Let the axis of rotation lie along the x axis, and at the instant of consideration let the canal be situated in the xy plane. Consider an element $efgh$ of the canal. The force due to an angular acceleration will be tangent to the circular path traced out by each such element. Since the plane of the circular path for any element of the canal is at right angles to the plane of the canal, there will be no component of this tangential force along the canal. There will therefore be no pressure change at B due to angular acceleration.

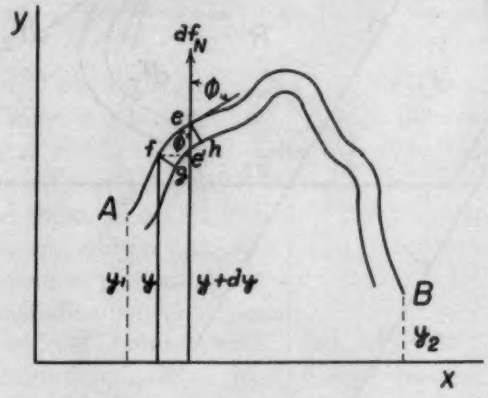

Figure 6

On each element dm there acts the centripetal force $df_N = dm\omega^2 y = \rho S dl \omega^2 y$, in which y is the radius of rotation of the element about the x axis and $ef = dl$ is the length of the element. The component of this force along the canal is $dF = df_N \cos\phi$. Making use of the relation $\cos\phi = \frac{ee'}{ef} = \frac{dy}{dl}$, we get $dF = \rho S \omega^2 y dy$.

Hence
$$p = \frac{F}{S} = \frac{1}{S}\int dF = \int_{y_1}^{y_2} \rho\omega^2 y dy$$

and
$$p = \rho\frac{\omega^2}{2}(y_2^2 - y_1^2). \qquad (8)$$

Now consider the case in which the axis of rotation is parallel to the plane of the canal but lies outside of the plane, as shown in figure 7. Take the x axis along the projection of the axis of rotation $O'C$ on MN, the plane of the canal. Let the y axis be as indicated in the diagram and the equation of the curve of the canal AB is $y = f(x)$. On rotation about the axis $O'C$ each element of the canal traces out a circular path

in a plane which is perpendicular to plane MN and parallel to the y axis. Consider the element $efgh$ of mass dm and length $ef = dl$ as shown in figure 7. Let d be the distance of the axis of rotation to plane MN. The radius of rotation R of any element dm is thus $R = \sqrt{y^2 + d^2}$. Acting on this element dm are two mutually perpendicular forces, $df_N = dm\omega^2 R$ and $df_T = dm\alpha R$, both of which lie in the plane of the circular path. Each of these forces can be resolved into components, one

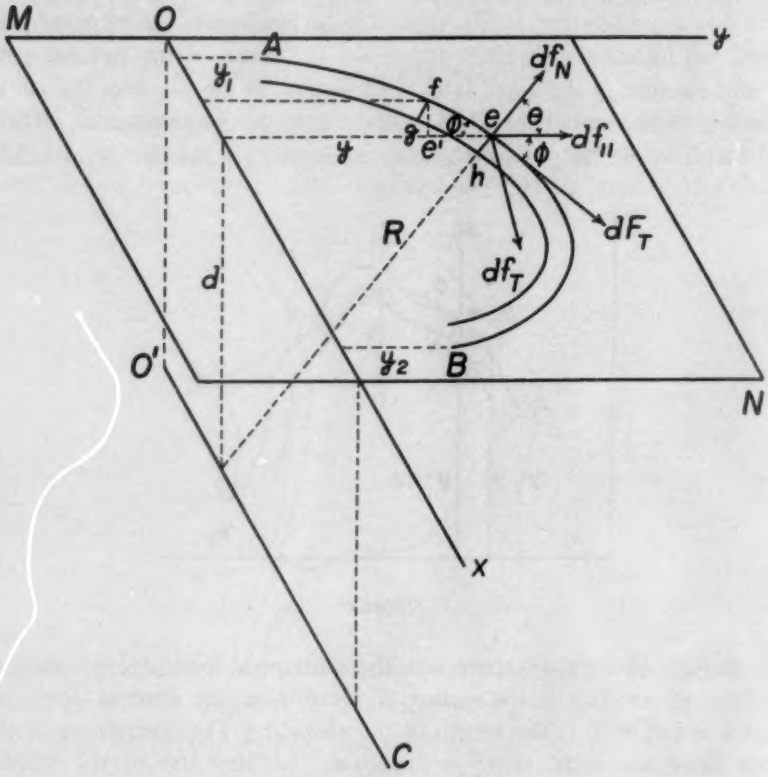

Figure 7

parallel and the other perpendicular to the MN plane. The component perpendicular to the plane of the canal, or the MN plane, will contribute nothing to the pressure difference between A and B and may be neglected.

If df_{11} be the sum of the two parallel components, then

$$df_{11} = df_N \cos\theta + df_T \sin\theta = dm\omega^2 R \cos\theta + dm\alpha R \sin\theta,$$

since $\cos\theta = \frac{y}{r}$ and $\sin\theta = \frac{d}{r}$, and we have $df_{11} = dm\,(\omega^2 y + \alpha d)$.

The force df_{11} has two components in the MN plane—one component, dF_N (not shown in the diagram), perpendicular to, and the other, dF_T,

tangent to, the canal. Since only the component along the canal contributes to the pressure increase at B, it alone will be considered. From figure 7 it can be seen that df_{11} lies in the xy plane and parallel to the y axis. Let ϕ be the angle that df_{11} makes with the tangent to the canal at the point e. Hence $dF_T = df_{11} \cos \phi = dm\ (\omega^2 y + ad) \frac{dy}{dl}$ since $\cos \phi = \frac{e\,e'}{ef} = \frac{dy}{dl}$. Putting in for the value of dm its equivalent, $\rho S dl$, we get the equation

$$dF_T = \rho S\ (\omega^2 y + ad)\ dy.$$

Changing this from force to pressure units, we have the equation

$$dp = \tfrac{1}{S}\ dF_T = \rho\ (\omega^2 r + ad)\ dy.$$

Integrating between the indicated limits we obtain the equation

$$p = \rho\omega^2 \int_{y_1}^{y_2} y dy + \rho ad \int_{y_1}^{y_2} dy = \rho \tfrac{\omega^2}{2} (y_2^2 - y_1^1) + \rho ad\ (y_2 - y_1). \quad (9)$$

In equation 9 when y_2 equals y_1 or when AB is parallel to the axis of rotation, the value of p becomes zero. On the other hand, when AB is perpendicular to and in the plane of the axis of rotation, the value of p is a maximum for a given angular velocity.

Finally, let the canal have a motion of pure translation. It is immediately evident that a linear acceleration perpendicular to the plane of the canal can produce no pressure difference between A and B and therefore under our assumption may be neglected.

Consider the case of an acceleration parallel to the plane of the canal. Let a, the acceleration, make an angle, θ, with the x axis (fig. 8). For convenience, the coordinate axes may be rotated through an angle, θ, so that the acceleration, a, will be parallel to the new axis, x' (fig. 9), and $y = f(x)$, the equation of the curve of the canal referred to the xy axes, becomes $y' = \phi\ (x')$ when referred to the $x'y'$ axes.

Consider the force on the element $efgh$ of length dl and mass $dm = \rho S dl$. The force exerted by the element on the canal is $dF = a dm$. The component of dF tangent to the canal is

$$dF_T = dF \cos \phi = dF \frac{dx'}{dl}$$

in which

$$\cos \theta = \frac{e\,e'}{ef} = \frac{dx'}{dl}.$$

Hence

$$dF_T = a\rho S dx'$$

and

$$dp = \tfrac{1}{S} dF_T = a\rho dx'.$$

Integrating,

$$p = a\rho \int_{x'_1}^{x'_2} dx'$$

and

$$p = a\rho\ (x_2' - x_1'). \quad (10)$$

When the acceleration is parallel to AB, the pressure difference between A and B is a maximum for a given acceleration, a.

From equations 8, 9 and 10 it is evident that the effect of a rotation about the axis OC (fig. 6) is equivalent to the effect of a rotation about an axis in the plane of the canal plus the effect of a linear acceleration, $a = ad$, in the plane of the canal.

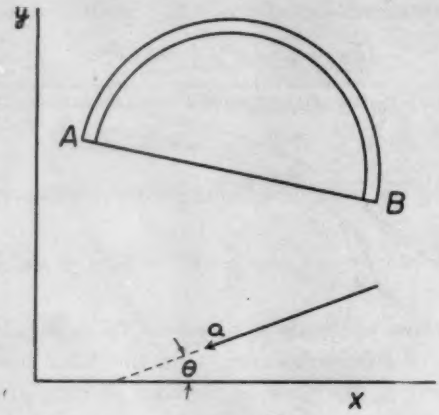

Figure 8

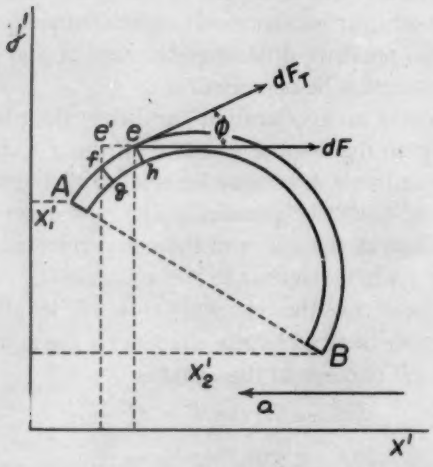

Figure 9

SUMMARY

The following cases for the motion of a canal have been considered:

1. Rotation about any axis perpendicular to the plane of the canal.
2. Rotation about any axis parallel to the canal.

3. Motion of pure translation in a direction perpendicular to the plane of the canal.
4. Motion of pure translation in a direction parallel to the plane of the canal.

Since any motion that a canal may have can be resolved into one or more of these cases, it is evident that every possible type of motion has been covered.

It has been shown that a hydrostatic pressure difference between the ends of a canal may be produced by angular acceleration or velocity, depending on the axis of rotation, or by linear acceleration.

The mathematical expressions for the hydrostatic pressure differences produced by the various motions are quite simple and would lend themselves very readily to experimental verification.

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TIC DOULOUREUX OF THE GLOSSO-PHARYNGEAL NERVE

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True glossopharyngeal neuralgia is extremely rare. Weisenburg [1] was the first to describe the symptoms of irritation of the glossopharyngeal nerve, although the symptoms he described occurred in a patient with a tumor of the cerebellopontile angle involving the tenth as well as the ninth nerve, and compressing the fifth nerve. The symptoms referable to the compression of the fifth nerve were relieved by removal of the gasserian ganglion. Pain, however, persisted and was recognized by Weisenburg as being in the domain of the glossopharyngeal nerve. The pain was at the root of the tongue, then was referred to the ear and down the neck and was associated with burning in the throat and a constant sense of dryness. The paroxysms occurred spontaneously or were brought on by eating. No motor weakness was noted.

In 1920 Sicard and Robineau [2] reported 3 cases of glossopharyngeal neuralgia though they considered that the vagus and the superior cervical sympathetic nerve were also involved and recommended extracranial section of the glossopharyngeal nerve, the pharyngeal branch of the vagus nerve and the superior cervical sympathetic nerve in order to relieve the pain, failing to recognize the condition as a distinct glossopharyngeal neuralgia. Since Weisenburg's first description,[1a] Doyle [3] has reported 4 cases, Adson [4] 5, Singleton [5] 3 and Dandy [6] 2. Although all of these

1. Weisenburg, T. H.: (*a*) Cerebello-Pontile Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, J. A. M. A. **54**:1600 (May 14) 1910; (*b*) The Diagnosis of Facial Pains from Those of Tic Douloureux and Their Treatment by Alcohol Injection, Therap. Gaz., Detroit **27**:305, 1911.

2. Sicard, R., and Robineau: Algie vélopharyngée essentielle, Traitement chirurgical, Rev. neurol. **36**:256, 1920.

3. Doyle, J. B.: A Study of Four Cases of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. **9**:34 (Jan.) 1923.

4. Adson, A. W.: Surgical Treatment of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. **12**:487 (Nov.) 1924.

5. Singleton, A. O.: Glossopharyngeal Neuralgia and Its Surgical Relief, Ann. Surg. **83**:338 (March) 1926.

6. Dandy, W. F.: Glossopharyngeal Neuralgia (Tic Douloureux), Arch. Surg. **15**:198 (Aug.) 1927.

cases were reported as instances of glossopharyngeal neuralgia, the doubt persisted that the pain was confined exclusively to the distribution of the glossopharyngeal nerve. For this reason, in the cases of Doyle [3] and Adson [4] the branches of the vagus, as well as the glossopharyngeal nerve itself, were divided. There seemed to be uncertainty as to the sensory nerve supply of the glossopharyngeal nerve in the pharynx.

Dandy [6] reported 2 cases in 1927 not only "to emphasize the clinical picture of this remarkable and not uncommon condition, but also to present a simple form of treatment" that was practically devoid of danger and productive of permanent cure without disability.

ANATOMY OF THE GLOSSOPHARYNGEAL NERVE

The anatomy and function of the glossopharyngeal nerve have been discussed exhaustively by Adson [4] and Doyle,[3] and their monographs should be carefully read by those interested in this subject. The glossopharyngeal nerve is a mixed nerve with both motor and sensory fibers, including those of the special sense of taste. The sensory fibers reach the mucous membrane of the middle ear, tongue, tonsillar fossa and oral pharynx. The nerve originates in the medulla oblongata and the anterior column of the cord, with its superficial origin from the rostral end of the posterolateral sulcus of the medulla oblongata in a line with the tenth and eleventh nerves. It has two ganglions, the petrous and the dorsal root ganglion. Leaving the skull through the jugular foramen, it passes between the internal carotid artery and the internal jugular vein and passes behind the styloid process. At the lower border of the stylopharyngeus muscle, it turns around and runs forward superficial to it. It breaks up into terminal branches which ascend to the palatine tonsil, the posterior third of the dorsum of the tongue, the soft palate and glossoepiglottic folds, and the anterior aspect of the epiglottis. Before reaching the side of the tongue it supplies the stylopharyngeus muscle and sends branches to the pharynx.[7]

The branches of the glossopharyngeal nerve are (1) tympanic, (2) pharyngeal, (3) muscular, (4) tonsillar and (5) lingual. The glossopharyngeal nerve with its branches connects intimately with the sympathetic system, as well as contributes to the geniculate and otic ganglions through the tympanic branch. It has two larger ganglions, the petrous and the dorsal root ganglion, at the lower and the upper part of the jugular foramen. Since the tympanic branch, as well as other connecting branches, arise from these ganglions, one naturally supposes that section of the nerve above these ganglions is required if the neuralgia is to be cured. If glossopharyngeal neuralgia is similar or analogous to trifacial

7. Jamieson, E. B.: A Companion to Manuals of Practical Anatomy, ed. 3, New York, Oxford University Press, 1932, p. 236.

neuralgia, this would seem to be true. On this assumption, Adson [4] formulated an operation for exposing and resecting the glossopharyngeal nerve through an intracranial approach.

CLINICAL FEATURES OF TIC DOULOUREUX

The close similarity between glossopharyngeal neuralgia and tic douloureux of trigeminal origin has been commented on by Sicard and Robineau,[2] Doyle,[3] Adson [4] and Dandy.[6] Both varieties of this fearful pain equally deserve the appellation of tic douloureux. The characteristic sudden ticlike paroxysms of excruciating pain and the induction of the pain by external stimuli are common to each.

In the approximately 30 cases of glossopharyngeal neuralgia assembled from the literature the clinical picture is strikingly uniform. Paroxysms of pain always strike some part of the glossopharyngeal sensory area—the tonsil, the back of the tongue or the pharynx. The pain is always unilateral and induced by liquids or solids passing over the sensory endings of the ninth nerve in the mouth or the pharynx. The pain is always excruciating; it is variously described as lancinating, knife-like or like stabs of a red hot iron. Usually each individual pain lasts only a few seconds, rarely over a minute; the duration of the paroxysm is therefore usually less than that of trigeminal neuralgia, but it would seem to be more severe, if that is possible. There is a strong tendency for the paroxysms to occur in a series over a period varying from a few days to a few weeks; in nearly every case intermissions of several months have occurred. The longest period of relief was three and three-fourths years in one of Adson's [4] cases, but the identical pain always returns in precisely the same location. In some cases there has been a tendency for the pain to shift from an initial point more remote and eventually to reach the central area of the glossopharyngeal sensory domain. In one of Adson's [4] cases it was in the lobule of the ear and the angle of the mandible, but the pains were brought on by swallowing or other stimuli of the tongue and pharynx. In nearly all cases pain radiates to the ear, the tongue and ear, or the tongue and pharynx. Not infrequently the pain begins there even before the more central pain in the intranasal area. Rarely does the pain radiate elsewhere than to the ear or its environs and to the affected side of the throat. Occasionally the ear is actually tender to touch and in a case of Adson's [4] touching the ear would precipitate the attacks. The method par excellence of starting attacks of glossopharyngeal neuralgia is to drink hot or cold liquids—cold is more effective than hot—but the attacks are also induced in many other ways, such as swallowing; solid foods seem to be less effective than liquids; talking, yawning, coughing, shouting, sneezing, touching the angle of the mandible—any of these may start an attack. Often the attacks seem to occur spontaneously.

TREATMENT

The injection of alcohol for tic douloureux of the glossopharyngeal nerve is not to be considered, because of the close proximity of the glossopharyngeal to the vagus nerve and large blood vessels. Therefore, surgical procedures must be resorted to.

External Approach for Extirpation of Glossopharyngeal Nerve in Neck.—The peripheral operation for exposure and section of the glossopharyngeal nerve in the neck was attempted by a number of operators. Adson [4] described an operation for the extracranial evulsion of the nerve which seemed to give relief over a long period but, according to the author, "it is a delicate procedure, complicated and difficult; necessitating gentle retraction of nerves." Singleton [5] reported 3 cases of cure by the external route, with some suggested variations in Adson's [4] procedure. The external operation is performed as follows:

An incision is made parallel to the anterior border of the sternocleidomastoid muscle, extending upward for a distance of 2.5 cm. above the tip of the mastoid and downward for a total distance of 10 cm., exposing the sternocleidomastoid. After retracting the muscle outward, the dissection is carried inward and upward behind the angle of the mandible and the body of the parotid gland. As the dissection is carried deeper, the posterior belly of the digastric muscle and the stylohyoid muscle come into view, crossing the operative field in a transverse direction. The posterior belly of the digastric is surprisingly large as compared with the anterior belly and partially covers the stylohyoid muscle. Both muscles are divided, and the ends of the muscles are attached to mattress sutures and retracted out of the field. Now the spinal accessory nerve comes into view. This passes obliquely downward and outward from the jugular foramen underneath the posterior belly of the digastric. The external carotid artery is gently retracted outward above the point where the occipital artery has been given off, at which point the hypoglossal nerve, which lies superficially and lateral to the jugular vein, is exposed. The facial nerve is not seen directly, as the operation proceeds above it, but its position must be constantly kept in mind. On dissecting free the jugular vein from its bed of reticular tissue, the vagus nerve is brought into view with a mesial pharyngeal branch, which is easily mistaken for the glossopharyngeal nerve. By carrying the dissection backward and upward to the jugular foramen, it can be identified. Carrying the dissection mesially, one encounters the internal carotid artery and the upper portion of the cervical sympathetic ganglion. Now the glossopharyngeal nerve will be located emerging from the jugular foramen closely anterior to the vagus nerve, external to the interior carotid arteries, and dipping sharply downward along the posterior belly of the stylopharyngeus muscle before it disappears in the muscles of the pharynx.

Intracranial Section of Glossopharyngeal Nerve.—Dandy [6] described intracranial division of the glossopharyngeal nerve in 1927.

The incision used is that used in performing unilateral cerebellar decompression; it is carried from the spine of the atlas upward to the external occipital protuberance; then, in the form of a horseshoe it is carried up to the lateral sinus and down over the mastoid process to its tip; the skin and muscles are

reflected in the same manner as they are reflected for cerebellar decompression, and the bone is removed upward and laterally, exposing the lateral and sigmoid sinuses. The bone is removed mesially and downward until the external occipital crest, the posterior condyloid foramen and the margin of the foramen magnum are approached; the dura is then incised and reflected mesially with a basal flap along the crest of the occipital bone. If the cerebellar lobe is not easily displaced, one can either drain the posterior cistern or the posterior horn of the lateral ventricle through a separate incision above the lateral sinus. Before elevating or displacing the cerebellar lobe, it is well to cover the cortex with strips of cotton; then with the illuminated retractor elevation of the lateral lobe of the cerebellum is accomplished, whereupon the seventh and eighth cranial nerves can readily be seen entering the internal auditory meatus. Inferior to these nerves and slightly more superficial, one will observe the ninth, tenth and eleventh nerves in a position at almost right angles from the medulla. It will be seen that the glossopharyngeal nerve enters the jugular foramen at the upper portion and is separated from the vagus by a small dural band, which is less than 1 mm. in width but definite enough to permit one to pass a small right angle ganglion knife between the fibers of the vagus and the glossopharyngeal nerve, thus facilitating a sharp section of the glossopharyngeal nerve.

HISTORY OF A CASE IN WHICH PERIPHERAL SECTION WAS DONE

V. H., a well developed, obese woman aged 67, had general arteriosclerosis and associated hypertension, for which she was retired from the Army Nurse Corps in 1935. Otherwise general physical examination gave negative results. In November 1938, at a luncheon party in Los Angeles the patient had a sharp stabbing pain in the region of the right tonsil lasting three to four minutes, which then recurred at intervals. The pain was so severe that she ran out of the dining room in great consternation. The pains continued day and night for six weeks. She described the pains as shooting, lancinating and stabbing; the attacks were induced by swallowing, by talking and by pressure in the right side of the neck. She was treated at this time by a physician in Los Angeles, who "cleared up the pus in her tonsils by application to her throat." The character of the application used is not known. There was no recurrence of these attacks for two years. On Nov. 15, 1940, while riding on a train from the East she thought she was "taking a cold" because of a peculiar dryness and tenseness in her throat. She suddenly experienced shooting pains in the right side of her pharynx and neck. These pains increased rapidly in severity. She recognized them as a duplication of her 1938 syndrome. They were not constant but occurred in paroxysms at frequent and irregular intervals. They were aggravated by coughing, talking and swallowing liquids or food. Pressure on the outside of her neck in the region of the tonsils would bring on a paroxysm of pain. In other words, there was a definite trigger action in these paroxysms. She used various home remedies, hoping that the pains and paroxysms would cease spontaneously, but without avail. On December 5 she was observed for the first time in the clinic for diseases of the eye, ear, nose and throat at the Letterman General Hospital and treated as an outpatient. The pain and distress became more severe each day. On December 27 she was admitted to the hospital with a diagnosis of glossopharyngeal neuralgia.

A survey in the hospital produced the following information: The blood pressure was 198 systolic and 100 diastolic; repeated examinations showed only minor variations. There were three nonvital teeth (the first canine teeth on the

left and the right and the second bicuspid on the right) with some roentgenographic evidence of change. The dental radiologist stated in his report: "These teeth do not seem to be involved in the present pathologic condition." The ear, nose and throat were negative for foci of infection. Two small tonsillar tags were present but were not considered to require surgical removal and were not believed to have any relation to her pain. The roentgen appearance of the bones of the skull was essentially normal. A roentgenogram of the cervical portion of the spinal column showed marked hypertrophic changes in the cervical vertebrae with considerable narrowing of the disks between the fifth and sixth vertebrae. However, the orthopedic surgeon stated that he did not believe there was any connection between this condition and the patient's symptoms. A roentgenogram of the chest showed that the aorta was slightly wider than normal, but there was no evidence of aneurysm. The heart was essentially normal. The urine was repeatedly negative for albumin and sugar. Its specific gravity averaged 1.018. The red blood cell count was 3,410,000; the white cell count, 6,800, with 62 per cent neutrophils. The hemoglobin content was 70 per cent. The Wassermann reaction of the blood was negative repeatedly. The blood showed urea nitrogen 15 mg. and sugar 106 mg. per hundred cubic centimeters. The electrocardiographic report showed slight slurring of QRS complexes. The T waves showed diphasia in lead III and a QRS complex in lead IV of varying amplitude, but in the electrocardiographic interpretation it was stated that there were "no significant abnormalities."

During her sojourn in the hospital she was given physical therapy and treated with salicylic acid, high voltage roentgen rays to the right side of her neck (a total of 1,200 roentgens being given), cobra venom, and vitamin B both by mouth and by vein. She was given large doses of a codeine salt repeatedly for the relief of pain. However, relief from the paroxysms could be obtained only by the application of a 5 per cent solution of cocaine hydrochloride to the right tonsillar area. Relief was prompt, but the drug had to be reapplied in one to two hours. This form of treatment was continued with reluctance.

While in the hospital she was seen in consultation by Dr. O. W. Jones Jr., on the staff of Dr. Howard Naffziger. Dr. Jones confirmed the diagnosis of tic douloureux of the right glossopharyngeal nerve and advised ameliorative rather than surgical treatment because of her advanced age, the cardiovascular syndrome and the arteriosclerosis. There was pitting edema of both her ankles, a history of air hunger on climbing steps and other signs of early cardiac decompensation. Dr. Jones felt that these signs, associated with hypertension (systolic blood pressure, 200), were very significant even though the roentgenogram of the chest showed no significant enlargement of the heart and the electrocardiogram was interpreted as essentially normal. The patient was discharged from the hospital on Feb. 13, 1941, moderately improved. She was given a bottle of the 5 per cent solution of cocaine and advised to use it sparingly. She was seen in the clinic at intervals as an outpatient. During this period of partial recession, she was conscious of the syndrome in her throat, which she described as a feeling of cotton in the retropharynx and she had considerable fear in swallowing and talking; but severe paroxysms occurred only infrequently.

On May 1 she was again seen in the clinic. The pains had increased in severity and in frequency. Conversation, swallowing liquids or pressure on the right side of her neck acted as a trigger to initiate the paroxysms. During attacks she would assume a characteristic posture with her head bent down-

ward on the right shoulder. When asked why she assumed this posture she said that it relieved somewhat the agony of the paroxysm on the muscles of the right side of the neck. If she was talking at the beginning of a paroxysm, she would stop in the middlle of the sentence and resume her conversation only on the completion of the attack. Each attack lasted twenty to forty seconds. She appeared to be in great agony and was again admitted to the hospital. The attacks were occurring on an average of once every four minutes. There was no lessening in the number or the severity of the paroxysms at night, and swallowing of liquids particularly had a trigger action. The patient appeared haggard, was tired from loss of sleep, had gradually lost weight and was a pitiful object. As time went on she refused to talk and was much averse to swallowing food; her mental outlook became more and more depressed.

On June 10 extirpation of the right glossopharyngeal nerve in the neck was done by the external route.

Operation.—Local anesthesia was induced with a 1 per cent solution of procaine hydrochloride and a 1 to 30,000 solution of epinephrine hydrochloride. In addition, a basic dose of avertin with amylene hydrate was given by rectum (basic dose, 55 mg. of avertin with amylene hydrate per kilogram). The patient tolerated the avertin with amylene hydrate poorly. Respirations were reduced to 10 per minute; the pulse became thready and difficult to count. Three 1 cc. doses of a 25 per cent solution of pyridine beta carboxylic acid diethylamide (coramine) were administered within a period of one-half hour before there was a return of the circulation to near normal.

The belly of the sternocleidomastoid muscle was not cut as suggested by Adson,[4] nor was it found necessary to ligate the occipital artery or the internal carotid artery. The "bottle neck" of the operative route is formed not by these structures but by the tip and body of the mastoid process on one side and the angle and ascending ramus of the mandible with the parotid gland on the other. In the recess below, near the base of the skull the various structures lie in a bed of loose reticular tissue and can be moved about easily after they are dissected free. This was startlingly true of the internal carotid artery and to a lesser degree of the internal jugular vein and of the vagus nerve and the spinal accessory nerve. These structures could therefore be easily moved out of the operative field, and this was done by gentle traction. Particular care was observed to limit the pull and pressure on the vagus nerve. Bleeding was practically absent after the superficial vessels of the cervical region had been ligated, except for some oozing following the section of the posterior belly of the digastric muscle and of the belly of the stylohyoid muscle, but this was controlled by suture. Considerable time was spent in definitely identifying the glossopharyngeal nerve. It was confused with the pharyngeal branch of the vagus nerve, as this branch is about the same size as the glossopharyngeal nerve and runs in about the same direction. Irritation of the pharyngeal branch of the vagus nerve produced an unpleasant sensation in the throat not unlike the paroxysms of pain she had experienced for months. Only when this branch was followed upward to the jugular foramen was it seen to unite with the vagus nerve just after the latter emerged from the foramen. The glossopharyngeal nerve was found emerging from the jugular foramen in an almost transverse direction between the internal jugular vein and the internal carotid artery and then sharply changing its course in a downward direction on the bed of the stylopharyngeus muscle and disappearing in the muscles of the pharynx. The nerve is surprisingly small. In this patient it was not larger

than a fine piece of cord. Pressure on the nerve produced the characteristic lancinating sharp pain from which this patient had been suffering and clinched the identification. The nerve was cut as far down in the pharynx as it could be followed before giving off its branches and at the jugular foramen, the segment having a total length of 7 cm. The wound was filled with sulfanilamide powder, a rubber dam drain was inserted and the wound closed with dermal sutures. Healing was by first intention.

Postoperative Course.—The patient's convalescence was somewhat stormy. There was considerable difficulty in swallowing solid foods, due to the anesthesia of the right side of the pharynx and the paresis of the muscles of deglutition on this side. The food would lie in a pocket in the right piriform sinus for one to two hours, after which she would regurgitate it. This unpleasant phenomenon gradually subsided. Severe laryngotracheitis with cough also developed, owing to food particles that found their way into the trachea, but this cleared gradually with the aid of hot steam inhalations and other local measures and with the return of deglutition to near normal. She was discharged from the hospital on July 25. There was complete relief from her paroxysms following surgical intervention.

MOTOR AND SENSORY EFFECTS OF SECTION

In this patient the motor changes as a result of the dissection of the glossopharyngeal nerve were inconsequential after the first month following the operation. The dorsal wall of the pharynx moved equally well on both sides. There was no deviation. Difficulty in swallowing liquids was small. There was considerable difficulty in swallowing solid food for one month, with some regurgitation, and occasionally food was drawn into the larynx and trachea, causing severe paroxysms of coughing. Elevation of the palate was equal on the two sides, but at rest the palate appeared to sag slightly. This suggests that the palatal muscles forming the posterior arch are supplied by the ninth nerve. Inasmuch as the difficulty in swallowing subsided entirely within six weeks after operation I feel that the severing of the posterior belly of the digastric muscle and the belly of the stylohyoid muscle was the major cause of the difficulty in swallowing and that as soon as healing had occurred in these muscles, swallowing returned to near normal. There was a certain amount of handling of the nerves in the wound in the effort to identify them. This was true of the vagus nerve and its pharyngeal branch particularly. It may be that some of the postoperative difficulty in swallowing was the result of trauma to this nerve.

The sensory changes after surgical trauma to the glossopharyngeal nerve were interesting. Anesthesia was found in the region of the nasal pharynx, the soft palate, the right side of the uvula, the tonsillar fossa and pillars and the base of the tongue—all on the right side. The loss of sensation in all but the tonsillar fossa, however, was relative. Firm pressure and extremes of heat and cold were felt there. In the tonsillar fossa there was no sensation whatever.

Alterations in taste are interesting in such cases. In Dandy's [6] 2 cases there was complete loss of taste in the posterior third of the tongue, presumably on the operated side. One of the patients noted no change in perception of taste in the anterior two thirds of the tongue. The other noted alteration to the degree that on the normal side all tastes were recognized more quickly and registered with greater intensity than on the side of the cut glossopharyngeal nerve. In the patient whose case is reported here, loss of taste was demonstrable in the posterior third of the tongue on the affected side but not in the anterior two thirds.

It is still too early to note this woman's relief as a permanent cure. There may be a recurrence of her symptoms in the future. Her case is presented as one in which the external operation was done because intracranial intervention was contraindicated. Six months after the operation the patient was examined and was radiantly happy. There had been no pain since the operation. Difficulty in swallowing had disappeared. An examination of the throat revealed that the areas of anesthesia in the retropharynx, uvula and piriform sinus had all returned to normal. There was still a droop of the soft palate on the right with loss of sensation in the tonsillar fossa, but these effects caused no unpleasant subjective symptoms. The patient had regained her weight and more.

SUMMARY

Tic douloureux is an extremely interesting disease entity. It is characterized by short paroxysms of sharp, shooting, lancinating pain of overwhelming intensity in the regions of the tonsil and the root of the tongue and referred to the regions of the ear. Talking, swallowing or touching the area produces the characteristic attacks by so-called trigger action. The attacks continue with varying frequency and intensity.

Intracranial section of the dorsal root of the glossopharyngeal nerve gives complete and permanent relief and is the operation of choice. When the intracranial approach is contraindicated, peripheral section of the nerve can be performed. It is complicated, difficult and delicate, but can be done.

The motor, sensory and sympathetic sequelae of the operation are minor. The subjective signs of section are anesthesia of the retropharynx and tonsil on the affected side and loss of taste in the posterior third of the tongue. There may also be motor interference with the swallowing of solids.

In patients with this condition in whom intracranial intervention is contraindicated because of marked hypertension or cardiovascular disease, section and extirpation of the glossopharyngeal nerve in the neck are suggested as a means of obtaining relief. The greatest difficulty of the operation is that of recognizing the glossopharyngeal nerve.

EFFICACY OF VASOCONSTRICTOR AGENTS IN THE OBSTRUCTED NOSE

A QUANTITATIVE EVALUATION

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During the past decade there has been a considerable difference of opinion as to the comparative efficiency of ephedrine and its synthetic derivatives in the treatment of nasal obstruction. Previous attempts to determine their specific action in the obstructed nose have not provided reliable data, largely because of the limitations of the methods employed. In 1936 Schur and I [1] first described a new method for the quantitative measurement of nasal patency. The patency of the nasal airway was easily and accurately determined in terms of resistance to nasal air flow.[2] This method was employed to evaluate the rate and degree of nasal shrinkage. The present study deals with the quantitative effects of ephedrine, cocaine and some proprietary synthetic preparations administered in different concentrations and forms on the erectile tissue in the pathologic nose.

APPARATUS AND PROCEDURE

The apparatus (fig. 1) consists of a nasal catheter (*F*), a flowmeter (*D*) and manometers (*E* and *V'*) connected in series with a convenient source of suction (*V*). The flowmeter serves to provide a constant rate of air flow through the nasal airway, measured in terms of liters per minute. The manometers determine the resistance to the nasal air flow produced in terms of millimeters of water. The inclined manometer (*E*) is used to measure the minor differences in resistance which are too small to register with the vertical manometer (*V'*). With the tongue held (as shown in fig. 1) and the subject breathing calmly through the mouth, the nasal catheter is inserted gently into the nostril and a constant rate of air flow of 12 liters per minute is drawn into the open mouth, up through the postnasal

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space and out through the nasal airway. The readings are first taken with the inclined manometer, and when the resistance obtained is beyond its range readings are taken with the vertical manometer.

In the present study, before the given drug was administered several repeated and alternating readings were taken of each nasal airway at approximately fifteen minute intervals for a period of one-half hour to one hour. These readings served as a basis for comparison of the experimental effects produced. The drugs were administered in solution by means of pressure nasal sprays, while the volatile synthetic preparation amphetamine (benzedrine) was given through an inhaler.

Fig. 1.—Apparatus for determining quantitative evaluation of vasoconstrictor agents in the nose.

Approximately 0.6 cc. of the shrinkage solution was sprayed gently into each nostril. The volatile drug was taken in two deep inhalations through each nasal airway.

Readings were compared with the degree of erectile tissue swelling found on inspection and with the degree of subjective nasal obstruction. After the administration of the drug, readings were taken at one minute intervals until the maximum effect was obtained and then repeated at fifteen to twenty minute intervals until the subjective symptoms of nasal obstruction first reappeared. The intensity and duration of shrinkage were evaluated in terms of (1) the time required for the maximum effect to take place, (2) the duration of the maximum effect and (3) the total duration of the effect.

CLINICAL AND EXPERIMENTAL DATA

The effect of the given drug was observed in nonobstructed and obstructed nasal airways in the same and in different subjects.[3] The criterion for the absence of nonobstruction was considered to be the lack of subjectively noticeable nasal blockage and of infection. Persons with physical abnormalties, such as enlarged turbinates, septal deviations and spurs, which appeared obstructive but did not produce any blockage were categorically included in the group without obstruction. The

TABLE 1.—*A Clinical Classification of the Study Material and Experiments*

	Number	Experiments
Patients without obstruction		
Normal	5	20
Chronic hypertrophic rhinitis (quiescent)	3	9
Vasomotor rhinitis (quiescent)	6	15
Vasomotor polypoid ethmoiditis (quiescent)	4	4
Total	18	48
Patients with obstruction		
Acute infection of the upper respiratory tract	9	11
Subacute infection of the upper respiratory tract	15	18
Chronic hypertrophic rhinitis (active)	6	10
Vasomotor rhinitis (active)	9	17
Vasomotor polypoid ethmoiditis (active)	6	12
Total	45	68

TABLE 2.—*Age and Sex Distribution in the Clinical Groups Studied*

	Age	Male	Female
Patients without obstruction	18-55	7	11
Patients with obstruction	14-70	17	28
Acute infection of the upper respiratory tract	18-49	0	9
Subacute infection of the upper respiratory tract	14-41	6	9
Chronic hypertrophic rhinitis (active)	18-48	3	3
Vasomotor rhinitis (active)	26-44	4	5
Vasomotor polypoid ethmoiditis (active)	39-70	4	2
Total		24	39

group with obstructed airways consisted of patients who had acute or subacute disease of the upper respiratory tract and patients with active chronic hypertrophic rhinitis, vasomotor rhinitis or vasomotor polypoid ethmoiditis. The majority of these were found to have septal deviations. In every case my clinical findings were checked with those of another examiner.

The clinical and experimental data are summarized in tables 1 and 2 according to the number of patients studied, experiments completed and age and sex distribution. The patients were under observation for several months.

3. The subjects were selected from those attending the outpatient nose and throat clinic of the New England Medical Center.

RESULTS

The experimental data are shown in tables 3 and 4 and figures 2 to 5 inclusive.

An analysis of the data for the group without obstruction (table 3; fig. 2) shows that the most rapid and prolonged degree of shrinkage

TABLE 3.—*A Comparison of the Average Initial Maximum, Maximum and Total Vasoconstrictor Effects in the Group Without Obstruction*

Drugs	Average Time Before Maximum Shrinkage Was Obtained, Min.	Average Duration of Maximum Shrinkage, Min.	Average Total Duration of Shrinkage, Min.
Cocaine hydrochloride 0.5%	3.0	60	190
Cocaine hydrochloride 1.0%	2.0	75	200
Ephedrine hydrochloride 0.5%	3.0	60	195
Ephedrine hydrochloride 1.0%	2.0	70	230
Cocaine hydrochloride-ephedrine hydrochloride 0.5%	3.0	65	190
Amphetamine (inhalant)	3.0	100	270
Neo-synephrin hydrochloride 0.25%	3.0	140	290

TABLE 4.—*A Comparison of the Average Initial Maximum, Maximum and Total Vasoconstrictor Effects in the Group With Obstruction*

	Average Time Before Maximum Shrinkage Was Obtained, Min.	Average Duration of Maximum Shrinkage, Min.	Average Total Duration of Shrinkage, Min.
Acute infection of upper respiratory tract			
Ephedrine hydrochloride 1.0%	6.0	30	155
Cocaine hydrochloride-ephedrine hydrochloride 0.5%	6.0	35	110
Amphetamine (inhalant)	25.0	15	85
Subacute infection of upper respiratory tract			
Cocaine hydrochloride 0.5%	6.0	30	130
Ephedrine hydrochloride 0.5%	6.0	40	120
Ephedrine hydrochloride 1.0%	4.0	45	155
Cocaine hydrochloride-ephedrine hydrochloride 0.5%	3.0	25	150
Amphetamine (inhalant)	5.0	60	165
Vasomotor rhinitis			
Cocaine hydrochloride 1.0%	15.0	30	95
Cocaine hydrochloride-ephedrine hydrochloride 0.5%	15.0	40	155
Amphetamine (inhalant)	3.0	100	180
Ephedrine hydrochloride 1.0% in oil	30.0	45	165
Ephedrine hydrochloride (isotonic)	3.0	65	175
Vasomotor polypoid ethmoiditis			
Cocaine hydrochloride 0.5%	5.0	10	90
Ephedrine hydrochloride 0.5%	18.0	18	105
Ephedrine hydrochloride 1.0%	5.0	40	111
Cocaine hydrochloride-ephedrine hydrochloride 0.5%	...	100	190
Amphetamine (inhalant)	6.0	65	85

was obtained with the synthetic derivatives of ephedrine, such as amphetamine and neo-synephrin hydrochloride 0.25 per cent. Of these two drugs, the action of neo-synephrin was considerably greater. Aqueous solutions of cocaine hydrochloride and ephedrine hydrochloride in 0.5 and 1 per cent strength respectively produced about the same rate and degree of shrinkage and were considerably less marked in their

effects than the synthetic drugs. However, 1 per cent ephedrine hydrochloride in aqueous solution produced a more intense shrinkage than a corresponding strength of cocaine hydrochloride (table 3).

In complete nasal obstruction due to acute infections of the upper respiratory tract (table 4; fig. 3) it was found that amphetamine proved far less effective than the single and combined aqueous forms of ephedrine hydrochloride and cocaine hydrochloride in 0.5 and 1 per cent concentrations. The most likely explanation for the diminished effect of amphetamine is the decreased permeability of the mucous membrane in the presence of an acute inflammatory exudative process, with the result that a volatile drug fails to stimulate the peripheral

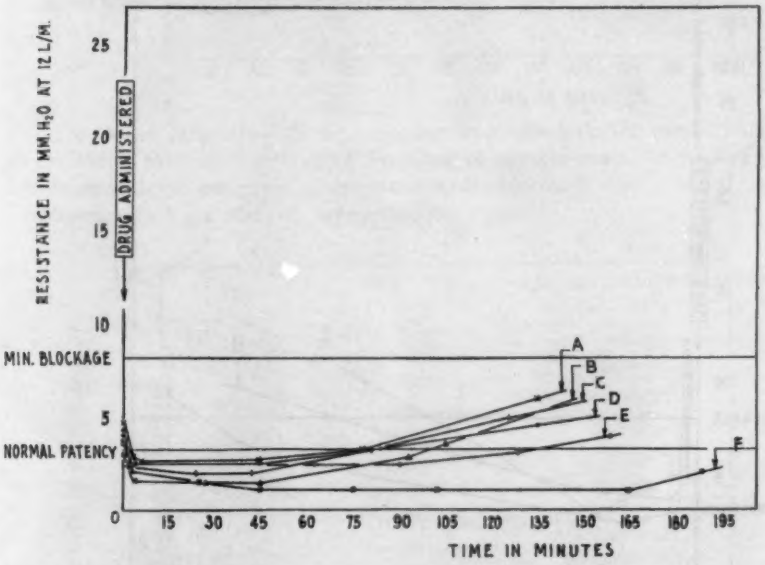

Fig. 2.—The comparative rate and degree of shrinkage in the nonobstructed nose. The line designated "normal patency" indicates the absence of subjectively noticeable nasal blockage and the degree of maximum shrinkage. The line labeled "minimum blockage" indicates the total duration of the effect produced. *A* indicates cocaine hydrochloride 0.5 per cent; *B*, cocaine hydrochloride–ephedrine hydrochloride 0.5 per cent; *C*, ephedrine hydrochloride 0.5 per cent; *D*, ephedrine hydrochloride 1 per cent; *E*, amphetamine; *F*, neo-synephrin in hydrochloride 0.25 per cent.

vasomotor mechanism with the same intensity as a freely miscible shrinking solution. It was further observed that a 1 per cent aqueous solution of ephedrine hydrochloride was much more effective than a combination of ephedrine hydrochloride and cocaine hydrochloride in 0.5 per cent strength.

In the subacute infections of the upper respiratory tract with partial nasal blockage (table 4; fig. 4) the intensity and duration of shrinkage caused by the drugs were found to parallel closely their effects in the normal nose. Here amphetamine was again observed to be more effective than either cocaine or ephedrine.

In vasomotor rhinitis with more or less complete nasal obstruction (table 4; fig. 5) the shrinkage produced by nasal vasoconstrictors resembled that noted in the subacute infections. Amphetamine was

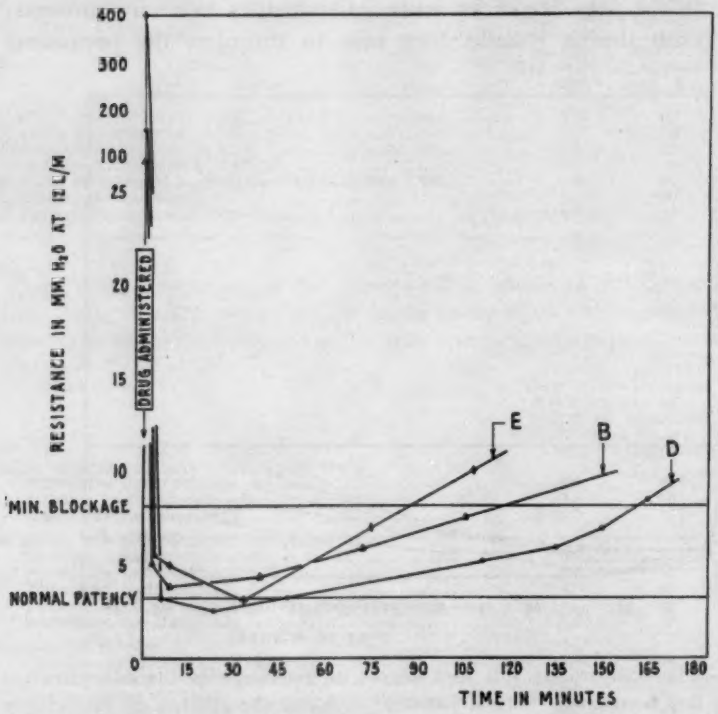

Fig. 3.—The comparative intensity and duration of shrinkage in acute infections of the upper respiratory tract with complete nasal blockage. *B* indicates cocaine hydrochloride–ephedrine hydrochloride 0.5 per cent; *D*, ephedrine hydrochloride 1 per cent; *E*, amphetamine.

more effective than aqueous or oily solutions of ephedrine and cocaine. However, isotonic solutions of ephedrine hydrochloride produced a more rapid action than 1 per cent ephedrine in oil, although the durations of their effects were about equal. One per cent cocaine hydrochloride appeared to be the least effective in its action, except when it was combined with ephedrine hydrochloride in 0.5 per cent concentration, which considerably increased its efficiency.

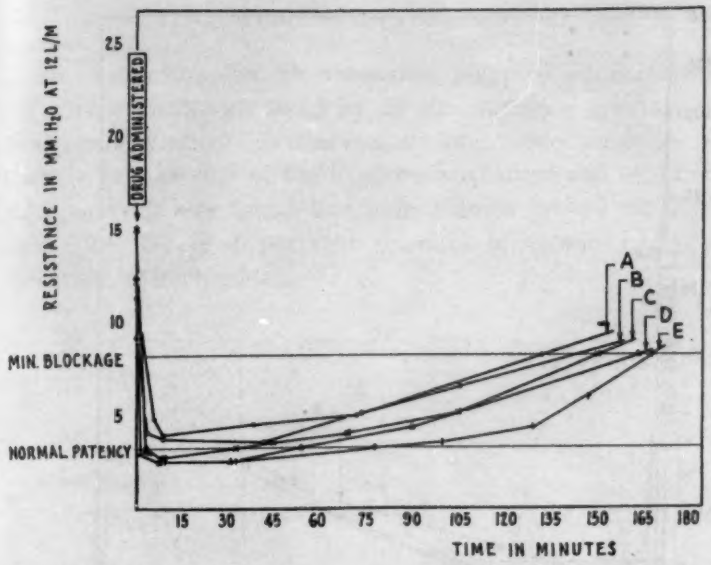

Fig. 4.—A comparison of the vasoconstrictor effects in the partially obstructed nose due to subacute infections of the upper respiratory tract. *A* indicates cocaine hydrochloride 0.5 per cent; *C*, ephedrine hydrochloride 0.5 per cent; *D*, ephedrine hydrochloride 1 per cent; *E*, amphetamine.

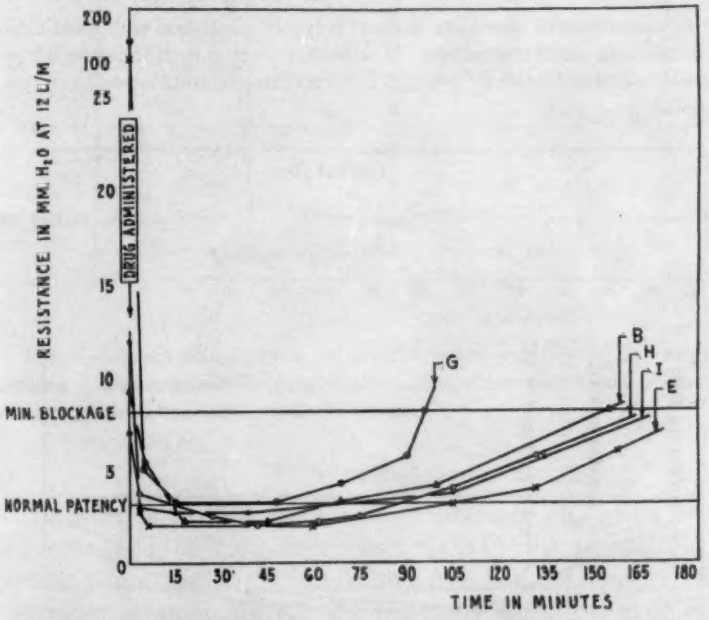

Fig. 5.—The shrinkage action of vasoconstrictors in the completely obstructed vasomotor nose. *B* indicates cocaine hydrochloride–ephedrine hydrochloride 0.5 per cent; *E*, amphetamine; *G*, cocaine hydrochloride 1 per cent; *H*, ephedrine 1 per cent oil solution (proprietary); *I*, ephedrine hydrochloride in isotonic solution (proprietary).

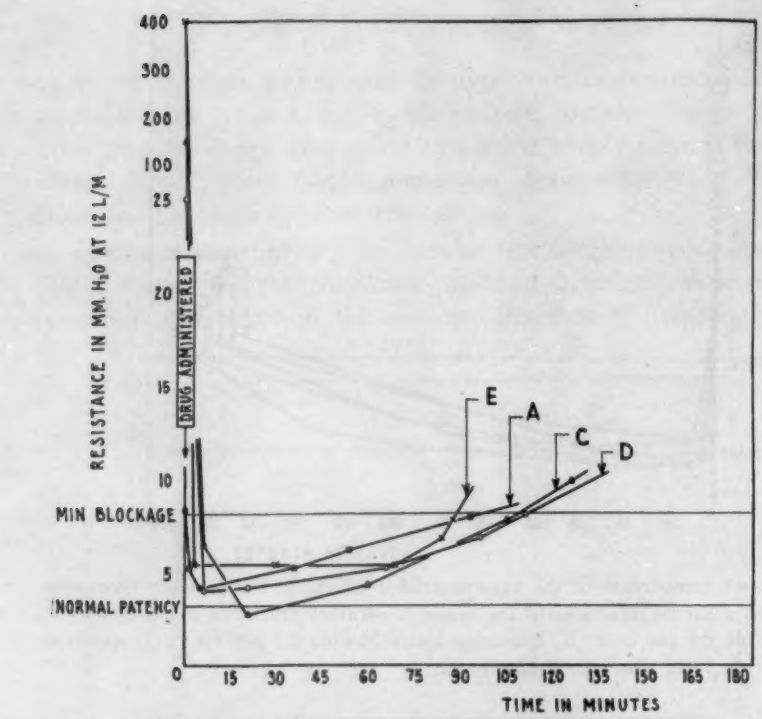

Fig. 6.—A comparison of shrinkage in nasal polypoid conditions with fixed tissue changes and complete nasal obstruction. *A* indicates cocaine hydrochloride 0.5 per cent; *C*, ephedrine hydrochloride 0.5 per cent; *D*, ephedrine hydrochloride 1 per cent; *E*, amphetamine.

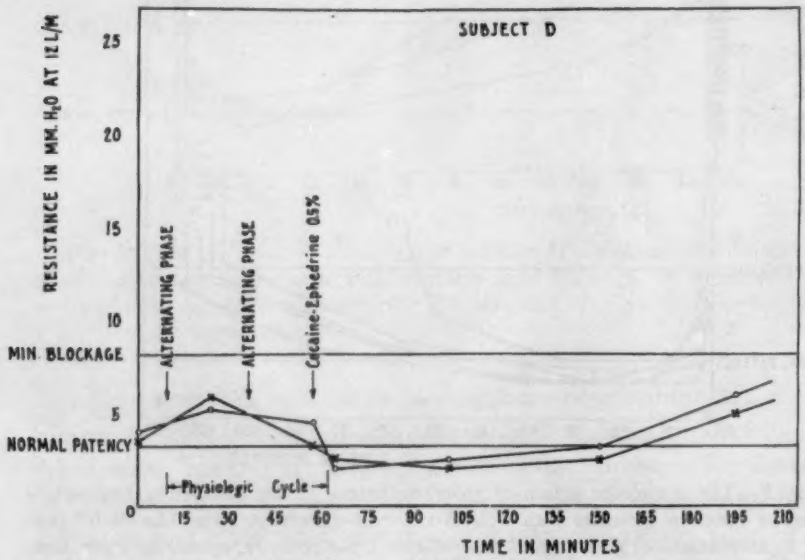

Fig. 7.—A physiologic cycle of erectile tissue swelling in a normal nose, showing a comparatively short alternating phase before and a prolonged phase after the administration of the drug.

In obstruction due to vasomotor polypoid ethmoiditis (table 4; fig. 6) the effects produced by all the shrinking agents were greatly diminished, even more so than in acute infections of the upper respiratory tract, largely because of the fixed tissue changes and secretion present. Here again it was found that amphetamine proved far less effective than either 0.5 or 1 per cent solutions of cocaine hydrochloride or ephedrine hydrochloride.

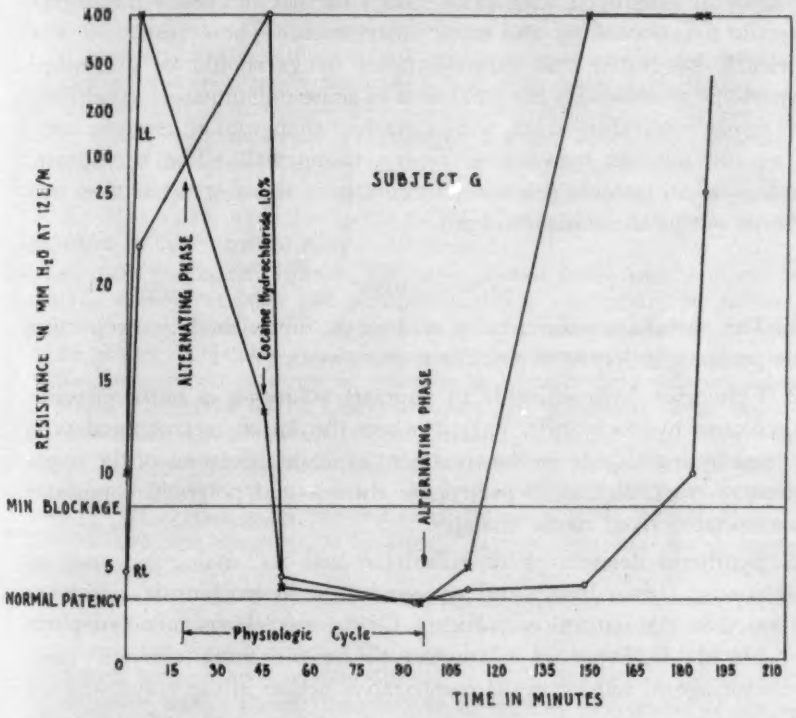

Fig. 8.—A physiologic cycle of erectile tissue swelling in an obstructed nose, showing a comparatively prolonged alternating phase before and a relatively short phase after the administration of the drug.

COMMENT

In order to evaluate the shrinkage efficiency of a given drug several factors should be considered, such as (1) the physiologic cycle of erectile tissue swelling (figs. 7 and 8), (2) the tonicity of a vasoconstrictor solution and (3) the means by which the drug is administered. It has been shown [4] by various observers that the physiologic

4. Uddströmer, M.: Nasal Respiration: A Critical Survey of Some Current Physiological and Clinical Aspects on the Respiratory Mechanism with a Description of a New Method of Diagnosis, Acta oto-laryng., 1940, supp. 42, p. 25.

cycle of erectile tissue swelling varies in point of time from one to four hours, with an average of two and a half hours, independent of position. The effect of a given drug, therefore, in different persons under the same conditions will vary according to the duration of the individual cycle present. In a previous study [5] it was shown that in the obstructed nose hypertonic solutions of sodium chloride greatly reduced erectile tissue swelling, while isotonic and hypotonic solutions tended to produce an increase in swelling and obstruction. It was also found that compressed air commonly employed with nasal sprays further increased the degree of erectile tissue swelling and nasal obstruction. These results, it was felt, clearly suggested that vasoconstrictor drugs should be prescribed in hypertonic solutions for the treatment of acute inflammatory conditions of the upper respiratory tract, with a gradual change to an isotonic solution as the mucous membranes return to normal. The therapeutic advantage of an isotonic solution particularly in the obstructed nose was not borne out by the evidence obtained.

CONCLUSIONS

1. The shrinkage efficiency of a drug is diminished in proportion to the pathologic degree of erectile tissue swelling.

2. Ephedrine hydrochloride in aqueous solutions is more effective than cocaine hydrochloride, except when the latter is combined with ephedrine hydrochloride, in the treatment of acute infections of the upper respiratory tract, chronic hypertrophic rhinitis and polypoid conditions with associated fixed tissue changes.

3. Synthetic derivatives of ephedrine and its analogues, such as amphetamine (benzedrine) and neo-synephrin hydrochloride, are more effective than the natural ephedrine. Of the two drugs, neo-synephrin hydrochloride 0.25 per cent appears to be the more efficient vasoconstrictor agent, judged by its comparative action in the nonobstructed nose.

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ABSTRACT OF DISCUSSION

DR. LEWIS T. BUCKMAN, Wilkes-Barre, Pa.: The results obtained by Dr. Sternstein's method of measuring the patency of the nose in terms of resistance to air flow indicate that gross structural findings do not give accurate information concerning the amount of nasal obstruction. He has divided his clinical material into a group with obstruction and a group without obstruction, the absence of obstruction being determined largely by lack of subjectively noticeable nasal blockage and of infection. Enlarged turbinates, septal deviations and spurs were found to be compatible with subjective breathing comfort. So also were chronic hyper-

5. Sternstein, H. J.: Quantitative Effects of Physical and Chemical Agents on the Erectile Tissue Response in the Nose, Arch. Otolaryng. **34**:532 (Sept.) 1941.

trophic rhinitis, vasomotor rhinitis and vasomotor polypoid ethmoiditis when they were quiescent. The group with obstruction was made up of patients with one of these three conditions when active and patients with acute or subacute infection of the upper respiratory tract. These patients presumably knew they had nasal obstruction because of subjective discomfort. In 1937 Dr. Sternstein proved that in terms of resistance to air flow through the nose the subjective symptoms were accurate criteria for determining obstruction. His present study deals with the quantitative effects of ephedrine, cocaine, amphetamine and neo-synephrin in the nose. In the nonobstructed nose, the effects of the several vasoconstricting drugs were not materially different one from the other except in the cases of amphetamine and neo-synephrin, with which the effects were more prolonged.

The effects of several strengths and combinations of cocaine and ephedrine and of amphetamine are reported, but I fail to find in the tables of results any reference to studies made with neo-synephrin in the obstructed nose. He describes the effect of neo-synephrin in the nonobstructed nose and in his final conclusion rates it higher in its effect than the ephedrine compounds and amphetamine. Since the thesis of this paper is a study of the obstructed nose, the dogmatic inclusion of this high rating of neo-synephrin in the nonobstructed nose with ignoring of its effect in the obstructed nose is unfortunate.

At first glance, the value of this paper seemed to lie largely in the critical analysis of different drugs used as vasoconstrictors. As pointed out earlier, however, an appreciation of the paper makes necessary a review of Dr. Sternstein's earlier works. This places the whole subject in a more advantageous perspective. It then becomes not so much an evaluation of vasoconstrictor effects of drugs as it does a contribution to the study of physics and physiology of the nose. In a previous study, Dr. Sternstein (ARCH. OTOLARYNG. **34**:523 [Sept.] 1941) demonstrated by means of this apparatus that air pressure commonly employed in the nasal spray produced no response in the erectile tissue of the nonobstructed nose but a prompt increase in the degree of nasal obstruction in the obstructed nose. Also demonstrated was the effect of hypertonic solutions to reduce the obstruction in the obstructed nose.

These earlier and the present interesting observations were made possible by the use of a mechanical apparatus and the living nose, the action of the vasomotor response being recorded. The method does not accurately represent natural conditions, because the nose piece fills the vestibule on one side and the vestibule on the other side is closed; the effect of the size of the opening of the nostril, the axis of its greater diameter and the directional effect of the air flow are all disregarded as they exist in the natural state.

One may speculate on what contributes more to loss of health and comfort: obstruction of the nose by infection or loss of ciliary action. One may speculate on what is more important to restoration of health and comfort: reduction of the obstruction or restoration and preservation of normal ciliary action. Given my choice, I should prefer the latter.

The ophthalmologist has discovered that the therapeutic effects of eye lotions sometimes are improved by a p_H variable from the normal p_H of the tears and that the successful use of contact lenses with hours of prolonged comfort often depends on the use of fluid between the cornea and the contact lens with a p_H variable from the normal p_H of the tears. Hypertonic solutions exert a beneficial effect on the swollen nasal mucosa, probably by inducing the expression of fluid in the tissue spaces outside the vessels and not by vasomotor control.

One reflects that whereas human beings may have originated in a saline menstruum compatible with sea water, their continued health and comfort may

not depend so stringently on surroundings isotonic with physiologic solution of sodium chloride.

Dr. John J. Shea, Memphis, Tenn.: I wish to report a clinical study made with a vasoconstrictor agent of the heptane group. In the second position amino was substituted, and a sulfate salt was the derivative studied. In 100 cases, equally divided between instances of allergic and of infectious conditions, 2 per cent ephedrine was used in one side of the nose and 2 per cent 2-aminoheptane in the other, and the latter was clinically more efficient. Not the elaborate method of estimating the capacity of the nose that Dr. Sternstein exhibited several years ago but the simpler office instrument, a respiratory mirror, was used. This simple metallic mirror is of value in office practice, for one cannot by inspection accurately estimate the respiratory capacity of the nose.

The 2-aminoheptane sulfate is slower than ephedrine in obtaining vasoconstriction but the effect lasts longer; the sulfate is equally efficient without causing any secondary symptoms. The action is purely a surface one, apparently without any accompanying constitutional symptoms. The after-reaction is very slight and compares favorably with that of ephedrine. It is not yet on the market but will eventually be, under the name "tuamine."

Dr. Herman J. Sternstein, Boston: In answer to the question raised by Dr. Buckman pertaining to the evaluation of neo-synephrin, I should like to point out that it is apparent from the parallel curves of amphetamine and neo-synephrin in figure 2 (the nonobstructed nose) and the general grouping in figure 4 (the partially obstructed nose) that these two synthetic drugs are about equally effective and are far more efficient in their action than either ephedrine or cocaine. It is the poor action of amphetamine observed in the acute condition, with complete obstruction, shown in figure 3, which leads one by strong inference to draw the conclusion that neo-synephrin is most probably the more efficient of the two and particularly the more desirable vasoconstrictor drug because of its miscibility.

I believe Dr. Buckman questioned the desirability of this method as a basis for establishing the functional patency of the nose by measurements taken of one side as compared with those of the other. Many of the patients with nasal respiratory difficulties rarely complain that both sides are obstructed except when the phases of a physiologic cycle happen to meet. While it is either one or the other side that is obstructed, it is their relative degree of obstruction as determined by this method that gives an accurate clue as to their total functional patency.

Case Reports

RHINITIS CASEOSA

J. Brown Farrior, M.D., Ann Arbor, Mich.

Rhinitis caseosa has been defined as a rare type of unilateral nasal disease characterized by the accumulation in the nose and paranasal sinuses of an extremely offensive, cheeselike mass, by the presence of a seropurulent nasal discharge and other manifestations of chronic suppurative sinusitis and, lastly, by intranasal, and frequently external, deformities which result from pressure exerted by the caseous mass.

REPORT OF CASE

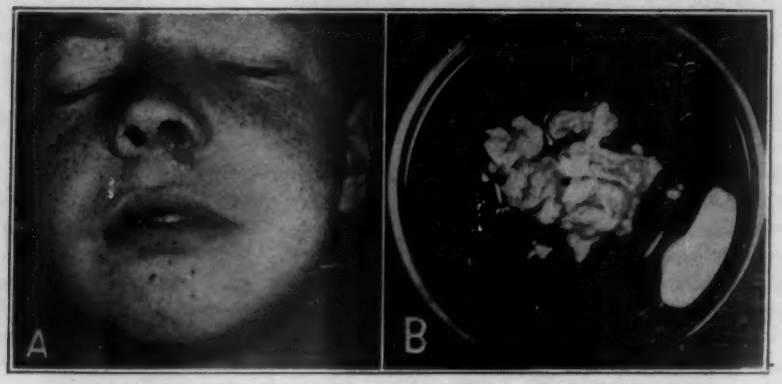
R. P., a 13 year old farmer's son from near Ionia, Mich., was admitted to the University Hospital on Nov. 28, 1939 with the complaint of complete nasal obstruction and copious foul nasal discharge. He had never been susceptible to colds and had had no nasal symptoms prior to the fall of 1938. At that time he had an acute infection of the upper respiratory tract, which was followed by a copious mucopurulent nasal discharge. The patient stated that he had used three handkerchiefs a day throughout the last school year. The nasal discharge remained mucopurulent for the first nine months of the disease, until the onset of the nasal obstruction in July 1939, when it became much thicker and was very offensive both to the patient and to his family. Occasionally a foul plug of grayish material came out of the right nostril, giving him relief from the nasal obstruction for the next two to three days. The obstruction had been progressive, becoming complete first on the right side, with secondary, slowly increasing blockage of the airway on the left. In the summer of 1939 a nasal polyp was removed from the right side. At no time during the present illness had there been excessive sneezing. There was no history of nosebleed. Three months before admission to University Hospital the patient first noticed the deformity of the right side of the nose and the right cheek.

The first examination disclosed the characteristic deformity of the face, that is, unilateral widening of the bridge of the nose, with the malformation extending toward the malar eminence. This area was not reddened. Palpation revealed no tenderness or crepitation of the underlying bony structures. Mucopurulent material was flowing from the reddened right anterior naris. The right nasal vestibule was completely filled with mucopurulent discharge. When the exudate was cleared away by means of suction, an underlying caseous mass was exposed. It was believed at the time that this mass covered a neoplasm. However, when a probe was passed into the caseous material it could easily be pushed backward into the nasopharynx, meeting with no obstruction and causing the patient no discomfort. Enough of the material was removed with the nasal forceps to establish a passage-

From the Department of Otolaryngology, University of Michigan Medical School.

Dr. Farrior is now serving as captain in the Medical Corps, University of Michigan Affiliated Unit, United States Army.

way into the nasopharynx. Through this the patient was given nasal irrigation, which brought forth about 1 ounce (28 Gm.) of very offensive, greenish gray, putty-like material. Subsequent examination of the right nasal chamber revealed that this mass had completely filled the cavity, producing pressure changes on all walls and in all directions. The nasal septum had been pushed completely over to the left side, resulting in almost complete obstruction of an otherwise normal left nasal chamber. The right inferior turbinate had been pushed downward against the lateral wall of the nose. The right middle turbinate was very small and was pasted against the lateral wall of the nose. It was covered with red, granulomatous mucous membrane. A polypoid mass hung from the anterior end of the right middle turbinate. Posteriorly, a mass of adenoid tissue could be seen in the nasopharynx. None of these structures bled during the manipulation or when palpated with a probe. The right frontal and right maxillary sinuses were dark on transillumination. Roentgenographic studies disclosed diffuse cloud-



A, facial deformity produced by the expanding mass within the nasal cavity; *B,* caseous mass after removal.

ing of all the paranasal sinuses on the right side. There was, however, no conclusive roentgenographic evidence of neoplasm.

At the time of the first examination biopsy of the polypoid mass on the anterior end of the right middle turbinate revealed inflammatory, edematous tissue, exhibiting infiltration with plasma cells and eosinophilic leukocytes. The section, however, did not resemble the nasal polyp found in cases of allergic rhinitis, as there was no increase in the number of goblet cells, the basement membrane was not widened and the infiltration of eosinophils was less than 10 per cent of the cellular element. A section of the caseous material revealed retained mucus, containing many bacteria.

During the first four days of hospitalization the patient was given frequent nasal irrigations of potassium permanganate. The offensive odor, which had been present for many months, completely disappeared in this brief period. The mucous membrane over the right middle turbinate lost its red, granulomatous appearance and became pale, so that it resembled early hyperplastic change. On December 1 the right maxillary sinus was given adequate intranasal drainage through a window in the inferior meatus. Probing through this window and irrigation of the right antrum disclosed no caseous mass and only moderate thickening of the lining

mucous membrane. Only mucopus was recovered in the antral washings. Culture of these washings produced a light growth of Streptococcus viridans, Staphylococcus albus and Micrococcus catarrhalis. Two biopsies at the time of the antrostomy revealed pronounced edema of the nasal mucous membrane and the characteristic changes of severe chronic suppurative rhinitis.

Further laboratory studies, including a blood count, complete urinalysis and a Kahn test of the blood, revealed nothing significant. A smear of material taken from the nose contained many spirochetes and fusiform bacilli. No acid-fast bacilli could be demonstrated. Cultures for fungi yielded no growth.

After the right antrostomy the patient was given daily irrigations of the antrum, with suction. The sinus infection subsided. Examination in April 1942 revealed no evidence of reformation of the caseous mass. The facial deformity was still present. Except for being slightly oversized, the appearance of the right nasal chamber was nearly normal. The septum was still greatly displaced to the left. The middle and inferior turbinates on the right side had returned to their proper positions, and their mucous membrane was regaining the smooth pink appearance. There was still some hyperplasia of the right middle meatus and the area of the anterior end of the middle turbinate. The lining mucous membrane of the right maxillary antrum showed considerable residual thickening. This rapid recovery, once the caseous mass has been removed, is one of the characteristics of rhinitis caseosa as it is presented in the literature; it demonstrates that radical operation on the sinuses is not indicated simply because there is diffuse clouding of all the paranasal sinuses and that these sinuses will clear of themselves if they are given an opportunity to drain properly.

COMMENT

Meyersburg, Bernstein and Mezz,[1] in their thorough study of rhinitis caseosa, presented the theories of the cause of this unusual malady.

1. Duplay, in 1874, conceived the idea that rhinitis caseosa develops after facial erysipelas which has extended to involve the nasal mucosa. Epithelial exfoliation and retention of the exfoliative debris result in the formation of the characteristic foul, cheeselike mass, which Duplay stated was analogous to the content of certain sebaceous cysts.

2. Cozzolini (1889) found an organism which resembled the tubercle bacillus.

3. Guarnaccia and others expressed the belief that rhinitis caseosa is a disease entity, of which Streptothrix alba is the causative organism.

4. Tillaux, Wagnier, Bruzzone and Otrich advanced the theory of "nasal cholesteatoma." Kelson, in 1904, provided evidence against the acceptance of this theory by his microscopic studies of tissues taken from the wall of the cavity after the removal of the cheesy mass. He observed the unchanged nasal columnar epithelium, surely not the kind that lends itself to the formation of cholesteatoma.

5. Hill and others stated the opinion that rhinitis caseosa is the result of a foreign body in the nose.

6. Bories and Schleicher stated that a necrobiotic change in one or several nasal polyps produced the putty-like mass.

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1. Meyersburg, H.; Bernstein, T., and Mezz, D.: Rhinitis Caseosa: An Analysis of the Literature and Report of a Case, Arch. Otolaryng. **23**:449-457 (April) 1936.

7. The predominantly accepted explanation is that rhinitis caseosa is a secondary condition symptomatic of an underlying primary nasal abnormality, such as sinusitis, polyp, neoplasm, septal deflection or other stenosing condition which tends to interfere with the egress of the discharge from the nose.

All of the aforesaid theories as to the cause of rhinitis caseosa have had their adherents. However, the last-mentioned one, that rhinitis caseosa develops secondary to some underlying abnormality, is accepted by far the greatest number of otolaryngologists.

Rhinitis caseosa is invariably a unilateral condition. S. L. Meyersburg described three clinical phases of the disease. The early stage is the period of unilateral (or bilateral) nasal discharge and other manifestations of chronic suppurative sinusitis. Examination reveals the ordinary signs of sinusitis plus some obstructive factor. In the second stage the discharge is more profuse and has an extremely offensive odor, both to the patient and to others. There is unilateral or bilateral nasal obstruction. Rhinoscopic examination then reveals the characteristic malodorous cheesy mass and the resulting pressure changes, as well as the remnants of the primary pathologic process. Other symptoms may develop from dacryocystitis and its complications. When the condition is neglected, the third stage, that of the facial deformity, develops; the lesion may progress and rupture externally, forming discharging bony fistulas.

According to all sources of information, the prognosis is excellent and the condition of the nose rapidly returns to normal, except for the permanent bony deformity, after removal of the caseous mass and the subsequent use of such ordinary cleansing measures as nasal irrigation. In the other 2 cases presented in the American literature [2] radical surgical intervention on the sinuses was performed, with exenteration of the necrotic ethmoid cells.

CONCLUSION

The history of this patient suggests that there first developed an ordinary acute infection of the upper respiratory tract, complicated by right maxillary sinusitis, which became chronic. The discharge from this suppurating sinus lodged in the right nasal chamber because of some mechanical factor or the lack of a sense of nasal cleanliness. As this purulent exudate stagnated in the nasal chamber, physiochemical changes took place, transforming the debris into the previously described disforming caseous mass. From the microscopic picture it appears that the nasal polyp developed secondary to lymph stasis in the anterior end of the middle turbinate, produced by pressure of the obstructing mass. Thus it would appear, that in this case, as in the one presented by Arslan,[3] rhinitis caseosa developed secondary to chronic suppurative sinusitis with stagnation and probably the growth of saprophytic spirochetes and fusiform bacilli in the stagnant mass.

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2. Otrich, G. C.: Cholesteatoma Involving the Ethmoidal Cells and the Antrum of Highmore, Illinois M. J. **48**:397-399, 1925.

3. Arslan, Y.: Ancora due parole sulla cosidetta rinite caseosa, Arch. ital. di otol. **5**:159, 1897.

TONSILLAR POLYP

GEORGE R. LAUB, M.D., CAMDEN, S. C.

Benign tumors of the tonsils are rare, only 150 cases having been reported prior to 1925, when Klestadt [1] made a study of such growths. Since then only a few cases have been added. It seems justified, therefore, to report a case of a benign polyp of the left tonsil.

REPORT OF A CASE

S. P., a Negress aged 59, came to the Johnson Memorial Hospital, Hemingway, S. C., with the complaint of difficulty in swallowing and talking for about a year. This condition had been becoming gradually worse during the last few months. Her medical history was without significance except for removal of a tumor of the left tonsil about fifteen years ago. No histologic examination was made at that time, and, owing to the death of her physician, I was not able to get any further information.

Physical Examination.—The physical condition was entirely normal except for a tumor, the size of an orange, protruding from the left tonsillar niche. It had the consistency of normal tonsillar tissue, but the color was a shade lighter. The tumor was movable, having a pedicle about 1.5 cm. long and 0.75 cm. wide. This pedicle was adherent to the lower pole of the tonsillar niche, probably in connection with some tissue from the lingual tonsil which was growing into the niche. No clinical signs of any inflammatory reaction were present.

The lymph nodes under the angle of the mandible and the superficial and deep jugular lymph nodes were enlarged, firm and indolent. The skin was not adherent to them, but it could not be made out with certainty whether or not they were attached to the deep tissues. Examination of the blood gave normal results, and the Wassermann reaction was negative.

Operation.—With the aid of local anesthesia, the tumor was peeled out of its bed very easily after ligation of several large blood vessels which supplied the growth. The pedicle was also removed without injuring any neighboring tissue.

The postoperative course was uneventful, and the swelling of the lymph nodes subsided in several weeks.

Pathologic Study (Dr. K. M. Lynch, per Dr. M. E. Cox, Medical College of the State of South Carolina, Charleston, S. C.).—The specimen received was an irregular, nodular, yellowish brown mass of tissue, measuring 5 by 4 by 3 cm., which had apparently been fixed in alcohol. The tissue was shrunken, as a result of fixation and loss of blood. The surface suggested the configuration

1. Klestadt, W.: Zur Kenntnis der gutartigen Tumoren der Gaumenmandel mit Bemerkungen über die Gaumenkapsel, Ztschr. f. Laryng., Rhin. **14**:47, 1925.

of a greatly hypertrophied tonsil (fig. 1). Section revealed rather soft, poorly preserved, grayish pink tissue, with a leafy arrangement.

The growth was of polypoid type and consisted of a dense, fibrous arborizing stalk stroma, covered by hyperplastic, chronically inflamed lymphoid tissue with fairly prominent follicles; the surface and the lining of the tonsillar crypts were usually stratified squamous epithelium. The surface epithelium and the tonsillar crypts showed heavy chronic and active inflammatory cell infiltrations. Beneath the latter, interposed between the surface epithelium and the lymphoid tissue, was an irregular zone of dense fibrous tissue, with a focus of fat tissue (figs. 2 and 3).

Diagnosis.—The diagnosis was tonsillar polyp.

Fig. 1.—Gross specimen of tonsillar polyp.

COMMENT

This case presented considerable diagnostic difficulty. The history of removal of a tumor of the tonsil fifteen years ago and the presence of a large mass, as well as hard, indolent, apparently fixed regional lymph nodes, in a woman of 59 were suggestive of a malignant growth or the recurrence of one. On the other hand, the appearance of the tumor, with the smooth surface, absence of ulcerations or superficial necrosis and lack of attachment to the neighboring tissues except by its pedicle, was against this possibility. Only a histologic examination could answer the question, and therefore the tumor was removed *in toto,* for the taking of a specimen for biopsy would have done much more harm than the complete removal of the tumor in case of a malignant growth.

In reviewing the literature only 1 similar case was found, that reported by New and Childrey [2] of a pedunculated tumor, 2 by 4 cm., which sprang from the region of the right lingual tonsil; histologically the mass proved to be chronically infected tonsillar tissue. In this case the patient was a

Fig. 2.—Section of tonsillar polyp, showing apparently normal tonsillar tissue and fat tissue. × 16.

2. New, G. B., and Childrey, J. H.: Tumor of Tonsil and Pharynx: Three Hundred Fifty-Seven Cases; Adenocarcinomas of Mixed Tumor Type; Seventy-Four Cases, Arch. Otolaryng. **14**:699 (Dec.) 1931.

15 year old girl, whose tonsils had been removed six years before. In all the other recorded cases no previous tonsillectomy had been performed. Friend [3] reported a case of a fibroadenolipoma of the tonsil arising from the cleft between the tonsil and the posterior pillar near the upper pole.

Fig. 3.—Section of tonsillar polyp. The cellular elements do not show any signs for malignancy. × 173.

3. Friend, L. J.: Fibro-Adenolipoma of the Tonsil, Arch. Otolaryng. 3:448 (May) 1926.

Evans and Odom [4] mentioned a case of polyp of the tonsil. Silbiger [5] reported 3 cases of small benign tumors—a fibroma, an accessory tonsil in the form of a papilloma and a polypous cyst. Kofler [6] demonstrated a large fibroma durum tonsillae before the laryngologic society of Vienna. Sturm [7] presented a case of a pedunculated tumor of the tonsil in a girl 11 years old, which histologically proved to be a fibroadenoma. Huenermann [8] brought excellent pictures of the different tumors which he had observed. He, as well as Klestadt,[1] discussed the classification of benign tumors of the tonsil.

Klestadt stated that he requires two questions to be answered in every case before classifying the tumor: First, is the mass a real tumor or only a hyperplastic, tumor-like formation, and, second, are the tissues derived from local tissues or from ones not normally present in this region? In my case it is not easy to answer the first question. The growth gave the appearance of a tumor, not only clinically but grossly, but the heavy chronic and active inflammatory cell infiltration, as well as the preservation of the structure, indicated an inflammatory process. It seems, therefore, that the inflammation was the primary cause of the development of this polyp. In regard to the second question, all the tissues of this tumor were derived from local ones, such as fibroid, lymphatic and fat tissue, Although it seems justifiable to classify this polyp as a benign tumor, the diagnosis of a hyperplastic, tumor-like formation seems to be most likely.

Although the morphologic classification of this growth gives some difficulty, a discussion of the cause and functional meaning is even harder. According to Borst,[9] a "defect always causes an increase in function as a reaction of the body, regardless of whether such a defect is mechanical or chemical." Likewise, an inflammatory reaction causes an increase in normal tissue and in all functions. The true tumor is a growth "antagonistic to the surrounding tissue and the whole organism." It is like a structure which does not fit into the plan of the organism. It has an independent, parasitic character.

Tonsillar tissue differs in its amount during the life of the individual; it is greatest in childhood and becomes more and more atrophic with advancing age. How can one explain the need of such enormous tonsillar tissue in a woman almost 60 years old? How does one account for the fact that no other part of Waldeyer's ring, or any other lymphatic tissue. was increased except the left lingual tonsil? Unable to answer such questions, one can say that functionally this polyp seemed to be "indepen-

4. Evans, W. H., and Odom, R. E.: Polyp of the Tonsil: Report of Cases, Ann. Otol., Rhin. & Laryng. **48**:495, 1939.

5. Silbiger, B.: Zur Kenntnis gutartiger Geschwülste der Tonsillen, Ztschr. f. Laryng., Rhin. **13**:217, 1924-1925.

6. Kofler: Fibroma Durum Tonsillae, Monatschr. f. Ohrenh. **56**:863, 1922.

7. Sturm, F. P.: Pedunculated Tumor of Tonsil, Brit. M. J. **1**:377, 1924.

8. Huenermann, T., in Denker A., and Kahler, O.: Handbuch der Hals-Nasen- Ohren-heilkunde, Berlin, Julius Springer, 1925, vol. 5, p. 313.

9. Borst, M., in Aschoff, L.: Pathologische Anatomie, ed. 7, Jena, Gustav Fischer, 1928, vol. 1, pp. 615 and 688.

dent and parasitic in character," a definition which leads one to assume that the growth was a real tumor in spite of apparent normal histologic structure.

Klestadt's anatomicohistologic classification, which disregards the functions of the tissue, seems not to be completely satisfactory. In consideration of these two differing points of view, it is not possible to make a definite statement about the nature of this polyp, which most likely is histologically a hyperplastic, tumor-like formation and functionally a true tumor.

526 DeKalb Street.

KERATOSIS OF THE LARYNX

H. B. Graham, M.D., San Francisco

It takes only a cursory review of the literature to reveal that not a large amount of research has been done on keratosis of the larynx. Possibly the reason for this is the scarcity of material and the ease with which most cases have been handled by removal of the lesion, together with its underlying structure.

The essential details of the observations that have been made may be summarized as follows: 1. Keratosis of the larynx is a precancerous disease. 2. The process is a localized cornification of the epithelium without invasion of the submucosa. 3 (to me most important). A chronic inflammatory process is diffused throughout the mucus membrane of the larynx, with localized areas of intensity about the base of the individual lesions. 4. The cause has not yet been determined.

Jackson and Jackson [1] made the statement that so far in no case had a malignant process developed, but New and Erich [2] reported that in 2 of 10 cases of keratosis of the larynx a squamous cell epithelioma grade 2 was present in situ and stated:

> In both leukoplakia and epithelial hyperplasia [often termed keratosis] there are marked subepithelial inflammatory changes. These conditions are considered together because they are intimately related to cancer. Epithelial hyperplasia is potentially malignant.

Morrison [3] reported a case in which an epidermoid carcinoma arose from a zone of keratosis in the subepithelial layer beneath the mucous glands but did not penetrate the underlying muscle. Finder [4] demonstrated a similar case before the Berlin Otolaryngological Society.

The very fact that the development of cancer in these precancerous lesions of the larynx is infrequent gives such lesions an added value in the study of the cause of cancer of the larynx in that there is not the incentive to act quickly and radically in their treatment.

To me the most important detail in the observations on keratosis of the larynx is the presence of a diffuse inflammatory lesion in the mucous membrane. If one could explain this phenomenon, it is more than probable that the underlying cause of the whole process could be determined.

Read at the Forty-Eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., June 2, 1942.

1. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 229.

2. New, G., and Erich, J. B.: Benign Tumors of the Larynx: Study of Seven Hundred and Twenty-Two Cases, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1938, pp. 17-96.

3. Morrison, L. F.: Tr. Pacific Coast Oto-Ophth. Soc. **25**:235-246, 1940.

4. Finder, G.: J. Laryng. & Otol. **40**:745, 1925.

In other parts of the body, such as the ear and nose, it is generally recognized that a persistent inflammatory lesion is frequently only local evidence of a fundamental chemical irregularity in the function of the body. Suppuration of the ear and mucous discharges, nasal catarrh, lymphoid hypertrophy, chronic colds, hypertrophy of the nasal mucous membranes, suppuration, etc., have been eradicated or greatly modified by attention to such factors as the diet and the endocrine glands, which simply means changing the chemical reaction of the tissues.

With these observations in mind, I wish to call attention to what has been to me an instructive case.

REPORT OF CASE

V. S., aged 66, in childhood had lived in Italy, where he had frequent sore throats. At the age of 14 he came to California and had no more trouble until 1933, when he gradually became hoarse. He was examined by a physician in 1938, who recognized keratosis of the larynx and referred him to me.

Family History.—The father was alcoholic; otherwise the family history was without significance.

Personal History.—The patient had smoked a pipe in 1938, but not since. He had taken considerable wine in early life. There was no history of venereal disease.

Examination and Operation.—The patient appeared to be in good health. The eyes, ears, nose and throat were normal except for the larynx, which showed reddening and thickening of the mucous membrane throughout, with areas of keratosis on the vocal cords. These areas appeared as white elevations, with the base redder than the surrounding tissue. The lesions were removed by the indirect method by means of a double curet, but recurred in two or three months, not necessarily in the same place. At times one cord would remain free for several months and then show new lesions. Fourteen biopsies were made over a period of four years.

Pathologic Study.—March 10, 1938: Histologic examination of tissue from the vocal cords revealed leukoplakia, with no evidence of malignancy. There was an area of marked epithelial hyperplasia and thickening, with hyperkeratosis. This lesion was not cancerous. One region of chronic inflammation with granulation and ulceration was noted.

April 28, 1939: The diagnosis was squamous cell carcinoma. The growth was largely warty and extended outward from the surface, with little penetration.

May 29: Biopsy of tissue from the esophageal surface of the left arytenoid cartilage revealed squamous cell carcinoma, consisting of a papillomatous growth of squamous epithelium with frequent mitoses. There was some irregularity in size and shape of the cells, as well as in the degree of differentiation.

August 4: An irregular, broad area of thickened mucosa with superficial piling up of the epithelium, suggested cornification. The base of the lesion contained irregular, metaplastic epithelium with occasional atypical mitoses. The basement membrane was still intact however. The diagnosis was squamous cell epithelioma of the larynx.

Jan. 26, 1940: The epithelium showed callous thickening but no evidence of malignancy. The epithelium of the mucous surface was greatly thickened and had a tendency to keratinization.

April 15: No evidence of carcinoma was noted.

June 25: Leukoplakia was present, with no evidence of carcinoma.

March 7, 1941: Leukoplakia, with early squamous cell carcinoma, was present.

Jan. 19; Feb. 16 and April 16, 1942: No evidence of carcinoma was noted.

It will be noted that on May 29, 1939 a well marked carcinoma occupied one third of the esophageal surface of the arytenoid cartilage. It was successfully removed by the indirect method by means of a double curet. The lesion was not preceded by any keratosis in this region but showed a tendency to development of a malignant process. On April 28 and Aug. 4, 1939 and on March 7, 1941 other specimens taken from the vocal cord showed an early squamous cell carcinoma

Fig. 1.—Squamous cell carcinoma on the esophageal surface of the arytenoid cartilage.

with the basement membrane intact. These are the only specimens that gave a positive picture of malignancy.

Laboratory Examination.—Feb. 28, 1941: The blood gave negative Kolmer and Kahn reactions. The urine was normal. Chemical studies of the blood revealed 10 mg. of calcium, 3.5 mg. of phosphorus, 18.9 mg. of potassium and 324 mg. of sodium per hundred cubic centimeters.

July 22, 1941: Studies of the blood again showed 10.1 mg. of calcium, 3.7 mg. of phosphorus, 19.6 mg. of potassium and 327 mg. of sodium per hundred cubic

centimeters. These values are all essentially within normal limits. The vitamin C content of the blood was 0.9 mg. (lower limit of normal). The vitamin B_1 output in the urine was 18 international units (in twenty-four hours), the lower limit of normal. Regenometric values [5] for vitamin A output gave 2.27 minutes for the first reading and 6.05 minutes for the second reading. The second reading normally should not be more than 2.30 minutes. This indicates either a low intake or poor absorption of vitamin A. No fungi were present in an implant from a biopsy specimen, and the growth was one of Staphylococcus aureus only.

Fig. 2.—Keratosis of the vocal cord with early carcinoma.

In 1941 the patient was placed on a diet rich in vitamins A and C and in the vitamin B complex, with a prompt reaction in the picture of the larynx. The red, inflammatory appearance of the mucous membranes changed gradually to the look of a normal larynx, leaving the halo about the keratotic lesions more pronounced; when the latter were removed there was not the previous tendency for the lesions

5. Blanchard, E. L., and Harper, H. A.: Measurement of Vitamin A Status of Young Adults by the Dark Adaptation Test, Arch. Int. Med. **66**:661-669 (Sept.) 1940.

to reform or for new ones to appear. At present the larynx is normal in general appearance; few small keratoses are present, and the voice, which formerly was husky, is clear.

The relationship between the quantitative and qualitative oral intake and the presence of cancer is being widely stressed by the medical and the lay press. Attention has been called to vitamin irregularities, the balancing of diets and the elimination of certain food chemicals, such as cystine. It appears that the attack on the cancer problem from this standpoint may throw some light on the baffling subject.

This present case is interesting not so much from the standpoint of the ultimate result, as the larynx is not yet entirely normal, but from the fact that a radical change in the general appearance of the mucous membrane appeared as soon as attention was given to the diet and that the keratotic lesions were greatly influenced by the treatment.

516 Sutter Street.

DESTRUCTIVE LESION IN THE PARANASAL SINUSES ASSOCIATED WITH BOECK'S SARCOID

JOHN E. BORDLEY, M.D., AND DONALD F. PROCTOR, M.D., BALTIMORE

Boeck's sarcoid, first described by Hutchinson in 1869 as a disease of the skin and later recognized by Boeck as a generalized disease affecting the lymph nodes and internal organs, has more recently been brought to the attention of the medical profession by the work of Longcope and Pierson.[1] In spite of the widened interest in sarcoid since that time, we have been unable to find any reference to its involvement of the sinuses. It is because of the apparent rarity of the condition and its interesting nature in this case that we make this report.

REPORT OF A CASE

At the end of October 1940, a 47 year old Negress, a widow, was admitted to the Baltimore City Hospital for the removal of an epulis in the region of the left canine fossa. A short time before admission her dentist had noticed this tumor in the gum and had advised its removal. There was a history of antisyphilitic therapy from 1936 to 1938 at another clinic. She had been suffering from fairly severe left frontal headaches for several weeks prior to admission, but except for this symptom and a slight dry cough she appeared in the best of health.

Physical examination showed mild cardiovascular hypertensive disease (with a blood pressure of 180 systolic and 110 diastolic and a slight systolic blow at the apex), the small tumor in the left canine fossa but no other abnormalities. The Wassermann reaction of her blood was negative; the precipitin test gave a positive result, the white cell count was 5,000 and the hemoglobin concentration 90 per cent.

The epulis was removed in the usual manner, and nothing extraordinary was discovered at the operation. Sections of the tissue removed showed the usual appearance of giant cell epulis.

At this time roentgenograms of the sinuses revealed "dense clouding of the left antrum and erosion of the left ethmoid cells and the left frontal sinus." A roentgenogram of the chest revealed "normal heart, slight dilatation of the aorta and patchy infiltration throughout both lung fields, due to metastases."

Examination of the nose and throat showed that the left antrum was dark on transillumination and the left middle turbinate thick and red. A little purulent discharge lay all along the left middle meatus.

General physical examination at this time revealed only the condition present on admission. A pyelogram showed nothing abnormal.

In the meantime the patient seemed well; the operative wound was healing; she still had headaches but complained little of them; there was little or no cough, and the temperature varied between 99 and 100 F. (rectally), rarely reaching as high as 101 F.

Two weeks after admission to the hospital, biopsy of tissue from the left middle turbinate, which by this time was very large and red, showed only signs of acute

1. Longcope, W. T., and Pierson, J. W.: Bull. Johns Hopkins Hosp. **60**:223, 1937.

and chronic inflammation but no malignant process. Culture of the pus which filled the middle meatus yielded Staphylococcus albus haemolyticus.

Two days later the anterior end of the left middle turbinate was removed and the anterior ethmoid cells were explored. The bone normally present in this region had been destroyed, and the area was filled with thick pus. Tissue removed at this operation showed only acute and chronic inflammation.

On Nov. 20, 1940 the patient was discharged from the hospital under potassium iodide therapy and was followed in the outpatient department. Her headache disappeared, and she continued to feel fine. The nose was treated with insufflations of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) powder, and the discharge greatly decreased. At this time it was felt that the most likely diagnosis was osteomyelitis of the sinuses on the left side, with destruction of the ethmoid cells, part of the frontal sinus and the antrum. The course had been unique for osteomyelitis in this region, with little fever and almost no symptoms, but no other explanation for the condition could be found. Healed miliary tuberculosis was considered a possible explanation of the appearance of the roentgenogram of the chest.

On Jan. 21, 1941 the patient was readmitted to the hospital. The white cell count at this time was 2,700 and the hemoglobin concentration 83 per cent. The Wassermann reaction was negative, but the precipitin and Eagle tests both gave positive results. The physical condition had remained unchanged since the previous admission. Two days later an ethmoidectomy was attempted on the left side. Where only two months before had been extensive destruction of bone, with a large pocket of pus, now almost the entire ethmoid region seemed filled with hard and sclerotic bone. At one place, a pocket of about 2 cc. of pus was opened, but elsewhere the bone could not even be dented. The mucous membrane removed at this operation showed on section groups of numerous small, hard tubercles. The pathologist suggested that this appearance was strongly suggestive of Boeck's sarcoid, if not tuberculosis.

The patient was discharged from the hospital on January 29 and has remained perfectly well.

After a diagnosis of Boeck's sarcoid was made, the patient was again studied thoroughly. No evidence of tuberculosis could be found. The tuberculin test gave a negative reaction to injection of tuberculin up to 1 mg., and studies of the chemical constituents of the blood revealed 25 mg. of nonprotein nitrogen, 7.63 Gm. of total serum protein and 3.41 Gm. of albumin per hundred cubic centimeters; an albumin-globulin ratio of 45 : 55, and 9.9 mg. of calcium and 3.2 mg. of phosphorus per hundred cubic centimeters.

Examination of the blood revealed: red cells, 4,400,000; hemoglobin, 11.6 Gm. per hundred cubic centimeters (80 per cent); volume of packed red cells, 39.0; white cells, 2,950, with 1 per cent juvenile forms, 64 per cent polymorphonuclear leukocytes, 8 per cent eosinophils, 1 per cent basophils, 19 per cent lymphocytes and 7 per cent monocytes. The sedimentation rate was forty-nine minutes, the icteric index 6, the mean corpuscular hemoglobin (concentrated) 30 micromicrograms, the mean corpuscular volume 89 cubic microns and the mean corpuscular hemoglobin (second determination) 26 micromicrograms. The platelet count was normal.

Biopsy of an axillary lymph node revealed "extreme hyperplasia of follicles in the lymph node, with 'tubercles' suggesting sarcoid." Roentgenograms of the hands and feet were normal.

Summary.—A 47 year old Negress who was operated on for epulis was incidentally observed to have lesions in the chest and sinuses (discovered roentgenographically) suggestive of a neoplasm. The lesions in the sinuses showed no evidence of malignancy on biopsy and were then thought to be due to osteomyelitis. Later, biopsy and subsequent evidence proved that the patient suffered from Boeck's sarcoid. At present (two years after the first admission) she is to all intents and purposes well.

6 East Eager Street.

Clinical Notes

COTTON SUTURE MATERIAL FOR TONSILLECTOMY

A. J. Podboy, M.D., York, Pa.

It is customary to use catgut for ligature and suture after tonsillectomy. Having observed the satisfactory use of plain cotton thread in general surgery and having read the articles by Meade and Ochsner and others,[1] I decided to try it after tonsillectomy because of its inexpensiveness, simplicity of sterilization, ease of handling and comparatively little danger of infection. The foreign body reaction due to this nonabsorbable suture is apparently of little consequence, since the cotton is probably eventually phagocytosed and digested.

"Cotton should be boiled 10 to 20 minutes and used wet. Cotton has been shown to decrease in tensile strength after autoclaving and increase after boiling provided it is tested while wet." [1b] "All contaminants were killed by boiling for twenty minutes or by autoclaving fifteen minutes." [1a] "Wounds sutured with cotton produced the least reaction and healed sooner [than with silk, linen or catgut]." [1b]

Unlike cotton, catgut becomes slippery when in contact with saliva and is sometimes difficult to thread in a small-eyed needle. The first knot can be tied more securely with cotton than with catgut and is not likely to slip.

Cotton can be wound on an instrument, boiled for ten to twenty minutes and kept wet with sterile saline solution.[1b] However, using it dry seems to have no great disadvantage in tonsil surgery. Clark's black thread no. 60 (125 yards [42 meters] for 5 cents) or finer thread may be used. "There is no need of using a suture which has greater tensile strength than the tissue in which it is placed." [1a] Surgeons have emphasized that knots are much better when tied with the fingers than with the biceps, to avoid strangulation of tissue. For more detailed information the aforementioned articles may be consulted.

Similarly, cotton thread can be used for suturing the incision after submucous resection when there is bleeding from the septal branch of the superior palatine artery or from the superior labial artery.

Since tonsillectomy is the most common surgical operation, constituting one third of all surgery, the convenience and economy would be considerable if cotton suture material were more generally used. This, of course, is of special importance in wartime economy. My experience with cotton suture material is limited. But I believe that any disadvantages which may be discovered when it is more generally used will be offset by the advantages. The sutures should be cut short, but occasionally the ends are found projecting into the tonsillar fossa a few days after operation. The sutures or ligatures can then be cut and removed.

211 South George Street.

1. (*a*) Meade, W. H., and Ochsner, A.: Spool Cotton as Suture Material, J. A. M. A. **113**:2230 (Dec. 16) 1936. (*b*) Localio, S. A., and Hinton, J. W.: Choice and Use of Cotton for Suture Material, Surg., Gynec. & Obst. **72**:615, (March) 1941.

NEW INSTRUMENTS

WILLIAM A. LELL, M.D., PHILADELPHIA

DECANNULATING CORKS FOR TRACHEOTOMY TUBES

Two types of corks designed for use in decannulating patients are described here. Both are of metal. One type is devised to use in the inner tube and is particularly adapted for patients who persist in having a moderate amount of

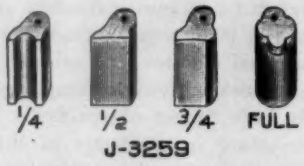

Fig. 1.—Decannulating cork for tracheotomy tube, type 1.

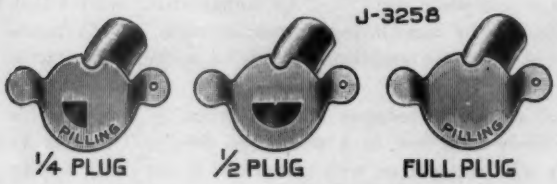

Fig. 2.—Decannulating cork for tracheotomy tube, type 2.

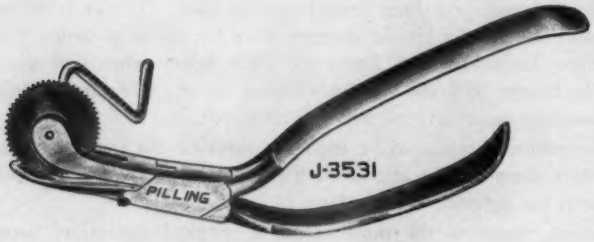

Fig. 3.—Laryngeal saw.

secretion in the tracheobronchial tree. Thus, the inner tube can be removed and cleaned when necessary. The cork is designed to fit the inner cannula of the tracheotomy tube and may be used as a quarter, half, three quarters or full cork (fig. 1).

The other type is designed to replace the inner cannula and is especially useful with patients who have little secretion. It also is made as a quarter, half or full plug (fig. 2). This decannulating cork may be obtained for no. 2 to no. 6 tracheotomy tubes. The particular advantage of this type of cork is that it can be kept in place much easier than a rubber cork. A special opening on the side to which a string may be attached keeps it from being misplaced or lost.

LARYNGEAL SAW

This saw was designed for use in cutting the thyroid cartilage in laryngofissure. The instrument is extremely useful in the operation in which the lesion is in the region of the anterior commissure, because the internal perichondrium can be protected after it has been separated from the cartilage. Thus it is possible to cut the thyroid cartilage without cutting the internal perichondrium. This eliminates cutting through the area of involvement. The saw may also be employed in separating the thyroid cartilage and the internal perichondrium in those cases in which the lesion is limited to either vocal cord.

1803 Pine Street.

CORRECTIONS

In the article by Drs. Schwartz and Isaacs entitled "Congenital Atresia of the Posterior Nares: Report of Two Cases," in the April issue (ARCH. OTOLARYNG. **35**:603, 1942), the sentence beginning at the bottom of page 605, referring to a report made by Dr. D. J. Morgenstern, is incorrect. In the article cited, Dr. Morgenstern stated that the obstruction was composed of bone, rather than being membranous.

In the article by Dr. J. Brown Farrior entitled "Contact Ulcer of the Larynx Developing After Intratracheal Anesthesia," in the August issue (ARCH. OTOLARYNG. **36**:238, 1942), the publisher of "The Larynx and Its Diseases," by Chevalier Jackson and Chevalier L. Jackson, was given as the W. B. Saunders Company. Credit should also be given to the Macmillan Company, which now publishes that book.

Abstracts from Current Literature

Ear

THE MIDDLE EAR AND ACCESSORY NASAL SINUSES FROM A FLIGHT SURGEON'S POINT OF VIEW. COLE D. PITTMAN, Ann. Otol., Rhin. & Laryng. **50**:518 (June) 1941.

Pittman points out the marked and sudden variations of pressure to which the sinuses and ears are subjected in the modern type of aerial warfare and expresses the opinion that prospective flyers should have careful examinations before acceptance in order to discover any pathologic condition in the nose or throat which might in any way interfere with proper functioning of these parts. Pilots with infections of the upper respiratory tract should be grounded until recovered. In the normal person aero-otitis media may be prevented by swallowing, yawning, shouting or contraction of the salpingopharyngeal muscles. However, in dive bombing none of these measures may work, and the pilot may have rupture of the drums. Treatment of aero-otitis media is outlined.

M. V. MILLER, Philadelphia.

OTOGENOUS TETANUS. SAMUEL ROSEN, Laryngoscope **51**:892 (Sept.) 1941.

Rosen reports a case of generalized tetanus complicating chronic purulent otitis media, with recovery of tetanus bacilli from the middle ear, indicating that the middle ear may be the focus of generalized tetanus.

A Negro aged 18 walked into the hospital, complaining of some pain in the right side of his face on chewing and of drooping of the right upper eyelid for four days. For the past two years his right ear had been discharging, and at times the discharge was foul. The temperature, pulse rate, respiratory rate and results of general physical examination were normal.

The right ear showed a large central perforation, through which a mass of red, swollen mucosa protruded and a moderate amount of mucopurulent material was discharged. There was no tenderness of the mastoid. The patient heard a moderately soft whisper. He had some weakness of the right side of the face. He could open his mouth to the limit, but on so doing complained of tightness in the region of the right mandibular joint. The following morning the facial weakness was more marked, and the patient could only half open his mouth. The next morning the patient was found in opisthotonos, with rigidity of the abdomen, some rigidity of the extremities and almost complete trismus. Generalized tetanic convulsions were precipitated by noise or movement. The spinal fluid was normal. Laryngeal spasm necessitated tracheotomy. The temperature rose to 104 F., and bilateral lobar pneumonia developed. Tetanus antitoxin was given intravenously, 100,000 units for two days and then 50,000 units for several days. This dose was later reduced to 20,000, 10,000 and, finally, to 5,000 units every three days. Altogether, 795,000 units of antitoxin was given within four weeks. At the end of two weeks the temperature became normal and the tetanic spasms almost entirely disappeared. The right ear discharged throughout, and the perforation became larger. Cultures of the discharge from the right ear showed Bacillus tetani and Bacillus proteus, confirmed by animal inoculation. Since the ear was considered the focus of tetanus, a mastoidectomy was done. The mastoid bone cut easily and was found to be diploic and soft. Cultures just before operation, at operation and since operation were all negative for tetanus bacilli. The patient recovered.

WOOD, Newark, N. J.

Pharynx

THE PAROTID GLAND. A. C. FURSTENBERG, J. A. M. A. **117**:1594-1598 (Nov. 8) 1941.

Furstenberg comments on the secretory glands that discharge their products into the oral cavity; after naming the parotid, submaxillary, and sublingual glands, the glands of Blandin and Nuhn, the palatal glands, the Moore glands and numerous small mucous glands scattered over the buccal mucous membrane and floor of the mouth, he limits himself to the parotid gland, emphasizing the anatomic, embryologic and certain clinical features. He discusses fully the infections of the parotid gland and their treatment and the tumors, with their clinical manifestations, pathology and prognosis. He concludes as follows:

1. Prominent among the causative factors responsible for inflammation of the parotid gland are foreign bodies in Stensen's duct, particularly calculus; injury or disease of the abdomen or pelvis, and extension of infection from neighboring tissues. Whatever the exciting cause of the abscess may be, dehydration and injury are potent influences in its development and progress. The role of the pharyngomaxillary fossa as a receptor and distributor of infections of the neck must be recognized because of its intimate contact with the inner prolongation (carotid lobe) of the parotid gland.

2. One cannot avoid the hard way of treating parotid abscess, namely, by wide incision which secures massive drainage. It is the most charitable method in that it minimizes suffering and hastens the patient's recovery. A practice that must find a permanent place in the treatment of infections of the parotid gland is the institution of thorough hydration. There is no compromise for this therapeutic measure.

3. Mixed tumors of the parotid gland usually appear during young adult life and are probably the result of some disturbance in the mechanics of embryonic growth.

4. A tumor of the parotid gland is vicious in its clinical manifestation, in that it causes little inconvenience until it has degenerated into a rapidly proliferating carcinoma. All early lesions probably contain malignant potencies.

5. The soft tumors are more prone to exhibit carcinomatous alterations than the hard, firm ones.

6. Surgical intervention which accomplishes the removal of every vestige of the lesion is usually indicated. Hope of a cure rests with the surgeon who performs the first operation. If he can successfully eradicate every remnant of the tumor, there is of course no danger of recurrence. When the lesion does recur, it usually presents frank carcinomatous alterations, and further surgical efforts are ineffectual.

7. The weight of clinical evidence is in favor of roentgen therapy after the extirpation of the neoplasm, particularly when there is reason to suspect that complete removal of the lesion has not been accomplished.

GORDON, Philadelphia.

POSTOPERATIVE HEMORRHAGES FROM THE TONSILS AND ADENOIDS. ANGEL BUSTILLOS and FRANZ C. JAHN, Bol. de los hosp. **38**:289 (June) 1939.

The authors discuss the etiologic factors that may predispose to post-tonsillectomy hemorrhages. They advise careful attention to prophylaxis and recommend that before operation patients be given calcium chloride supplemented by vitamin C. Liver extract, given both orally and parenterally, should also be used. They recommend that operation be postponed in the presence of certain conditions, namely, the residual period of an acute infection of the upper respiratory tract; menstruation; sudden changes in atmospheric conditions; active therapy, particularly the arsenic and bismuth treatment of syphilis; hemorrhagic diathesis, and secondary anemia.

If hemorrhage should occur, they recommend first the use of tampons saturated with one of the various coagulants. This is particularly valuable in the presence of bleeding from the adenoids. Here the postnasal pack should be inserted. In the tonsillar fossae ligation of the bleeding area is indicated. Transfusions, either autogenous or homogenous, are necessary in cases of the continuous or profuse type of hemorrhage.

PERSKY, Philadelphia.

TONSILLECTOMY IN CHILDREN. ERWIN SCHIFF, Pract. oto-rhino-laryng. **3**:358, 1941.

Schiff, in a detailed discussion of the pros and cons of indiscriminate and generalized tonsillectomy in children, takes the stand that this operation has been performed too frequently. After citing the observations of several investigators, particularly Kaiser, he expresses the belief that tonsillectomy is indicated only in selected cases. His indications are (1) frequent attacks of angina and peritonsillar abscess, and (2) pronounced and permanent hypertrophy of the tonsils, which, though they do not act as a focus of infection, interfere with the proper feeding of the child.

Tonsillectomy for the sake of removal of a focus of infection is definitely contraindicated, since there is no substantial evidence that the tonsils can produce focal symptoms. Here he is citing Reimann. He believes that the danger of pulmonary abscess or hemorrhage, the bad effect of narcosis and the greater liability of catarrhal inflammation of the respiratory tract in tonsillectomized children are sufficient to lead the rhinologist to exercise greater forbearance in the general recommendation of this operation.

PERSKY, Philadelphia.

Larynx

PHARYNGEAL AND LARYNGOPHARYNGEAL CANCER; A NEW COMBINATION OF SURGERY AND RADIATION THERAPY IN THEIR TREATMENT: PRELIMINARY REPORT OF FOUR CASES. MILLARD F. ARBUCKLE and A. C. STUTSMAN, South. M. J. **34**:219 (Feb.) 1941.

The authors call attention to the fact that the treatment of pharyngeal and of laryngopharyngeal cancer is unsatisfactory and state that because of the widespread ramifications of such neoplasms attempts at complete surgical removal have been universally disappointing. Treatment by irradiation has been more encouraging in so far as the tumor is concerned, but the results have often been marred by the destruction of normal tissue within the line of fire, resulting in infection and death of the patient from sepsis and hemorrhage. The process starts with injury to the laryngeal cartilages from the roentgen rays and by erosion spreads to the wall of the pharynx. In an effort to avoid injury to the laryngeal cartilages, the authors have removed the normal thyroid and epiglottic cartilages before starting roentgen therapy. Four patients have been treated by this method, with encouraging results. Each case is reported in some detail.

SINGLETON, Dallas, Texas.

TREATMENT OF LARYNGEAL PAPILLOMA IN CHILDREN. E. N. BROYLES, South. M. J. **34**:239 (March) 1941.

The author reports 5 cases of papilloma of the larynx in children. The condition cleared up in about six months when treated with local applications of estrogen. An estimated 1,000 international units (0.1 cc. of amniotin in oil) was applied at each treatment. No secondary sex changes were noted.

Many kinds of treatment have been used for papilloma of the larynx: excision, roentgen or radium irradiation, fulguration, local applications of chemical caustics and subcutaneous injection of testosterone. None of these have been satisfactory.

The author believes that the local application of estrogen has a definite action on the multiple papillomas of children. The characteristics of growth were changed from the infantile to the adult type, which made the treatment simple and far more satisfactory than treatments used in the past.

SCHLUTZ, Chicago [AM. J. DIS. CHILD.].

TREATMENT OF CANCER OF THE LARYNX. CHEVALIER L. JACKSON, South. M. J. **34**:243 (March) 1941.

In the treatment of cancer of the larynx, the author stresses the importance of early and accurate diagnosis and states that the diagnosis should always be confirmed by biopsy. He discusses briefly the advantage of irradiation and surgical procedures in the treatment of cancer of the larynx and states: "The relative radiosensitivity of different tumors as determined by their histopathology has been emphasized by some, and discounted by others, but most are agreed that a more important consideration in the selection of treatment is the gross location and extent of the lesion." In cases of cancer of the vocal cords the relatively high operability and curability are due to the fact that the tumor frequently begins on the true vocal cords, a region poor in lymphatic glands and one from which metastasis is late.

The technics of laryngofissure, laryngectomy and irradiation are described in some detail. Laryngofissure is the treatment of choice for cancer in the "anterior intrinsic area" of the vocal cords, and the operation should result in over 80 per cent of cures in suitable cases. Laryngectomy is indicated when the lesion involves the arytenoid region, with impaired motility, or when it extends into the subglottic region. The narrow field type of operation is advised. The operation should give at least 50 to 60 per cent of cures in cases of advanced intrinsic cancer and should have a low operative mortality.

Irradiation by the fractional (Coutard) technic or by radium bomb may be used when laryngofissure and laryngectomy are contraindicated.

The histopathologic changes should be studied and recorded. The prognosis is better in cases of tumors of the well differentiated, "low grade" type, whether treated surgically or by irradiation, but surgical treatment should be the first choice provided there is no contraindication.

SINGLETON, Dallas, Texas.

CAVERNOUS HEMANGIOMA OF THE LARYNX. ARISTIDES MONTEIRO, Acta med. Rio de Janeiro **6**:263 (Nov.) 1940.

The author reports a case of cavernous hemangioma of the larynx about the size of a bean which was expelled during a coughing spell. The disease apparently lasted about five years, and the tumor was found on the anterior commissure. Owing to the size and apparent interference with respiration, tracheotomy was considered, but during the examination the patient began to cough forcibly and brought up the tumor mass. There was little bleeding. The cords returned to their normal appearance, and the patient was completely cured when examined a year later. Histologic examination revealed a cavernous hemangioma.

PERSKY, Philadelphia.

OPERATIVE TREATMENT OF LARYNGEAL CARCINOMA IN CASES OF LATENT SYPHILIS. A. CAMPIAN, Monatschr. f. Ohrenh. **75**:205 (April) 1941.

In general, a positive Wassermann reaction is a contraindication to surgical intervention. The author operated on 2 patients, whose wounds healed without complication. He attributes this success partly to the intense antisyphilitic treatment given before and after operation and the regular use of potassium iodide.

LEDERER, Chicago.

LARYNGECTOMY. J. C. GROS, Pract. oto-rhino-laryng. **3**:369, 1941.

The author reviews a series of 24 cases of laryngectomy, in 15 of which he supplemented surgical treatment with irradiation. Of the 24 patients, 11 are living, 1 disappeared from observation and 12 died. There were recurrences in 9 of 20 cases in which operation was successfully performed.

In summarizing his data, he expresses the belief that laryngectomy is extremely delicate and fraught with risk in patients over 70 years of age. A preliminary tracheotomy eliminates the possibility of bronchopneumonia as a postoperative complication. When the tumor is seen roentgenographically to extend down to the level of the subglottic region, the first tracheal ring must be included in the extirpation. When metastasis has extended to the cervical lymph nodes, surgical treatment is the preferable procedure. Postoperative radiotherapy is indicated to reduce the frequency of recurrence and usually has no effect on the healing of the operative region. Five years of good health after the operation is no indication of protection or immunity from recurrence. PERSKY, Philadelphia.

Nose

CHOLESTEATOMA OF THE MAXILLARY, SPHENOID AND ETHMOID SINUSES. ARISTIDES MONTEIRO, Acta med., Rio de Janeiro **5**:13 (Jan.) 1940.

Monteiro described a case of a young man who had pansinusitus, for which he had had two previous operations. Owing to recurrence of his symptoms a Caldwell-Luc operation was performed, which revealed an extensive cholesteatomatous mass filling the maxillary, sphenoid and ethmoid sinuses. The floor of the nasal fossa was destroyed, but the frontal bone and sinus were not involved. After the operation there was complete cure. PERSKY, Philadelphia.

SCLEROMA IN SLOVAKIA. M. SIVAK, Monatschr. f. Ohrenh. **75**:183 (April) 1941.

From an epidemiologic point of view the appearance of scleroma in families is of importance. It is essential that in manifest atrophic catarrhal conditions the possibility of scleroma be borne in mind. Women show a greater affinity for the hyperplastic forms of the disease than men. In a survey of families, it was found that at least one parent was affected in three fourths of all families examined and both parents in one third. Nearly 50 per cent of the children showed pathologic changes, such as atrophy or a positive serum reaction, or were carriers of the bacillus. No doubt economic and hygienic conditions are of greatest importance. These were found to be bad in three fourths of the families, and these families also showed a higher incidence of sick members.

World War I is accused of having spread the disease into regions where it had been unknown before, chiefly through prisoners. The change in climatic and hygienic conditions seems to further healing, while recurrences can be observed after a return to the previous poor surroundings. A review of the literature is presented, and the following conclusions are drawn:

Scleroma is a chronic infectious disease of social and familial contagious character, probably produced by Frisch's bacillus. Through extraneous influences, primary bacillus-negative and seronegative catarrhal conditions, even familial atrophic rhinitis, with dry mucus and crust formation, may develop. In such surroundings Frisch's bacillus can act specifically, and the secondary atrophic seropositive form or the typical hyperplastic type of scleroma develops. Both these types may appear endemic. For the latter form, a certain intensity and duration of the infection, as well as poor hygienic conditions, seem essential.

The disease appears in early childhood, with a predilection for the female. It is contagious and is found in families. The two forms of the disease are pathologically opposed. If a low grade infection has allowed the body sufficient time for immunization, an atrophic change will appear; otherwise a hyperplastic

scleroma will be formed. Atrophies in the later age groups should therefore tend to eliminate the hyperplastic form. However, atrophic forms are less liable to be annoying and may remain undetected; therefore, cases of this type do not appear in statistical reports unless the entire family is routinely examined. The serum reaction (complement fixation) should be undertaken in every case of atrophic rhinitis. A survey of the distribution shows scleroma to be prevalent in regions with an abundance of fresh water.

LEDERER, Chicago.

LEIOMYOMA OF THE NASAL VESTIBULE. H. MERLIN, Monatschr. f. Ohrenh. **75**:202 (April) 1941.

Four months after an injury to the nose a tumor of the right nasal vestibular region developed in a girl of 18. The tumor rested on the pyriform crest and nearly occluded the nostril. On removal the tumor was found to be smooth and the size of a small egg; histologic examination showed it to be a leiomyoma. The origin of this tumor may be in mesodermal cells which have been torn from their connections during the development of the facial processes in an early embryonic period. A later traumatic influence may cause the growth of this congenital fissural tumor in the region of the union of the medial nasal process, on one side, and the lateral nasal process and the maxillary process, on the other.

LEDERER, Chicago.

MUCOCELE OF THE MAXILLARY SINUS, WITH OCULAR SYMPTOMS. A. FRANCESCHETTI and R. GUYOT, Pract. oto-rhino-laryng. **3**:329, 1941.

The authors reported the case of a man aged 29 who had complained of hyperplastic pansinusitis with recurrent polyposis since the age of 14. He was subjected to a series of operations over a period of years. These included several polypectomies, a bilateral Caldwell-Luc operation and a bilateral ethmoidectomy. After this last operation and institution of continuous medical supervision, he showed considerable improvement and remained apparently well for about six years.

However, at the time of admission to the hospital he complained of pain and swelling at the inferior margin of the right orbit. Since there was no pathologic condition of the nose other than a small synechia between the inferior turbinate and the spur of the septum, he was discharged and was not seen again for eight months, when he returned with evidence of a marked increase in the size and sensitivity of the infraorbital swelling. The swelling now involved the infraorbital region, was soft to the touch and rather sensitive. Roentgenographic examination revealed a soft tissue mass, with erosion of the inferior orbital plate. The ocular movements were not altered, nor was there any evidence of exophthalmos. An incision was made through the lower lid, and a large cyst full of greenish yellow mucoid material was removed. Subsequently a secondary Caldwell-Luc operation was performed on the right side, and here, too, the sinus was observed to contain the same type of secretion as was found in the cyst. Six months after the second operation the patient was discharged as completely cured. The pathologic examination confirmed the diagnosis of typical mucocele.

PERSKY, Philadelphia.

Miscellaneous

DISEASES OF THE RESPIRATORY TRACT AND AIR CONDITIONING. CAREY P. MCCORD, J. A. M. A. **116**:1360 (March 29) 1941.

The author discusses the various means of air conditioning, air purification, exhausts, et cetera, and cites the findings of some of the workers who have undertaken a study of this subject.

He justifies the following terms: (1) ventilation, (2) complete air conditioning, (3) partial air conditioning, (4) air conditioning for human beings, (5) air conditioning for wares and materials and (6) therapeutic air conditioning.

Several reports have already been published by industrial plants, and these vary in certain conclusions. Several of the larger plants claimed that "only the scantiest of differences have occurred as to respiratory disease frequency and severity." One large plant stated that they found "no evidence to indicate any improvement in incidence or duration of sickness among a group of upward of one thousand people working in scientifically controlled air conditioned space as compared with a control group of approximately the same size working in non-air conditioned quarters, ventilated by the usual mechanical and natural methods."

Phases of shock and vasomotor crisis concerned with air conditioning are discussed.

The author concludes with the following comment: (*a*) It will be impossible to place in the hands of the engineers any bill of air-conditioning particulars suitable to all persons uniformly because there exist so many different requirements for comfort and well-being among human beings. (*b*) Too much is expected of air conditioning. There will always be some persons who may require adjustment. (*c*) No comment has been directed to efficiency, comfort or noise abatement. Many forms of special therapies are rightfully esteemed, especially those dealing with modified atmospheric changes. (*d*) The closest cooperation among physicians everywhere, public health workers, hygienists, engineers and others will help to remove all possible physiologic stumbling blocks that hamper the application of air conditioning. (*e*) "All considered, at the present time, it appears that the control of ordinary respiratory diseases depends to a greater extent on 'human conditioning' than on air conditioning."

GORDON, Philadelphia.

ONE STAGE OPERATION FOR CLOSURE OF LARGE DEFECTS OF LOWER LIP AND CHIN. H. MAY, Surg., Gynec. & Obst. **73**:236 (Aug.) 1941.

Defects resulting from the removal of more than two thirds of the lower lip are usually closed by pedicle flaps from distant points, involving much time and several operations. Transplantation of local flaps can, however, be used. The author's operation consists of excision of the tumor in a heart-shaped piece. The incisions on either side meet below the center of the mandible. Flaps are outlined by an incision extending from the corner of the mouth upward to about 1½ inch (3.8 cm.) in front of the tragus of the ear and then carried down at less than a right angle, ending below the mandible. From the corner of the mouth the incision includes skin and muscles as far as the anterior border of the masseter muscle. Posterior to this it includes the skin and the subcutaneous tissue only. A flap of mucous membrane is raised on the inner aspect to turn outward as the future lining of the lip. Mobilization is carried out by incision of the mucosa along the labiogingival sulcus and a deep vertical incision through the mucosa and muscle along the anterior border of the masseter muscle. The defect created by the shifting of the flaps to the midline is closed by turning a muscle flap into the defect from the anterior part of the masseter muscle. Complete closure may then be readily carried out.

HENRY A. MILLER, Philadelphia.

FACIAL NEURALGIA OF DENTAL ORIGIN. ARISTIDES MONTEIRO, Acta med., Rio de Janeiro **5**:161 (March) 1940.

The author discusses the case of a patient who complained of intensive attacks of facial neuralgia on the right side. Every attempt to determine the etiologic factor and every type of treatment, even the injection of alcohol into the gasserian ganglion, was without any effect on the symptoms. Finally a canine tooth was removed, after which the symptoms cleared up.

PERSKY, Philadelphia.

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

WILLIAM E. GROVE, *Editor of Abstracts*

Sixty-Fourth Annual Congress, Atlantic City, N. J., May 25-27, 1942

(Concluded from Page 617)

Veins of the Pharynx. DR. O. V. BATSON, Philadelphia.

This article was published in the August 1942 issue of the ARCHIVES, page 212.

DISCUSSION

DR. JOHN F. BARNHILL, Miami, Fla.: The time has come when the anatomist who teaches the ear, nose and throat specialists must be a good otolaryngologist. To have the ordinary anatomist, who knows his anatomy as Dr. Batson says it was known up to 1871, teach these men now does not get them anywhere. Otolaryngologists need to correlate anatomic knowledge with everyday practice. All over this country courses in anatomy are being given, and in some instances, at any rate, general anatomists are teaching otolaryngologists. I do not believe that is a wise practice.

There should be in every school teaching such postgraduate courses a man who stands at the forefront of otolaryngology and who has the ability to use that special anatomic knowledge. Unless that is done, not much progress will be made.

For instance, for an anatomist to say that the external wall of the superior constrictor muscle is covered by a plexus of veins does not mean anything special to you or to me, but it is of value to have some one who has had great experience with the lesions of those veins tell the student why he needs to know about them. The general anatomist does not know the clinical application.

Dr. Iglauer, how is your course taught?

DR. SAMUEL IGLAUER, Cincinnati: Two laryngologists are employed to teach the anatomy. The general anatomist teaches the blood and nerve supply, but not the specific details.

DR. JOHN F. BARNHILL, Miami, Fla.: For instance, if in these courses the vagus nerve, with its branches to the sympathetic system, is described, the student does not get much out of it, but if some one who has had great experience in the function of the nerve tells him about its reflexes, he wants to know more about it.

DR. SAMUEL IGLAUER, Cincinnati: I should like to ask Dr. Batson why he had to tie off any veins. Was it to prevent the spread of the injecting fluid into some territory he was not investigating? I do not understand why he had to tie off a vein, which in itself produces, so to speak, an artefact.

DR. GORDON BERRY, Worcester, Mass.: May I ask Dr. Batson whether the veins which he described are within easy reach of the local anesthetics which are employed in operative work about the tonsils? I judge that they are posterior to the posterior and anterior pillars of the palate, in which injection is made.

DR. HARRIS P. MOSHER, Marblehead, Mass.: I heartily agree with what has just been said about the teaching of anatomy to otolaryngologists. I feel that only one who knows the applied anatomy can do this teaching. That has been my practice so far as I have been able to carry it out.

DR. O. V. BATSON, Philadelphia: There has been too much of a tendency to include facts in anatomy which point up the erudition of a teacher and which are not of any great significance at the moment.

I get out a letter now and then which I wrote a few years ago to some one starting the course, in which I suggested that his anatomy should be that of the best current clinical practice and that he should not try to anticipate what might be useful in 1952, but leave that to the men in 1952 to find out. I think one can show all the points in theoretic anatomy which one wishes with the use of practical examples.

As to Dr. Iglauer's question why it was necessary to ligate the superior laryngeal veins, I am not sure that it was, but let me consider the relationship. I injected from the dorsum of the tongue, from which the outflow is by way of the superior laryngeal vein. This vessel is the most ready outflow because it is larger and the communications go down into the anterior venous plexus. So by ligating the superior laryngeal vein I duplicated much the same condition that is produced every time one holds one's breath and strains. I increased the pressure in the superior laryngeal vein and then allowed the annulated plexus in the region to fill up. I think that is physiologic. Elze depended either on forcing the specimen or on securing one in which the valve was not competent; so he injected backward through the laryngeal vein. In at least half of my injections I was following the normal course of the blood. I do not think that is abnormal.

As to the problem of local anesthesia, I injected here the veins of the posterior pillar and those of the tonsil, and it is quite possible, I believe, to inject directly into one of these vessels, although they, for the most part, are very fine. The lymphatics of the region, which duplicate the course, although not the size, of the veins, could also, by inference, be injected directly. As a matter of fact, I have some specimens covering this same area.

Use of Roentgenograms of the Larynx. DR. FREDERICK M. LAW, New York.

Conventional roentgenograms of the larynx are not entirely satisfactory. It is impossible to obtain anteroposterior views, owing to the superimposition of the spine. Most reliance should be placed on the fluoroscopic examination, which must be made during and without phonation, preferably in the presence of the laryngologist.

The newer technic of the body section roentgenogram is the only method by which the subglottic extension of the lesion can be secured. This shows a cross section of the larynx at any predetermined depth and excludes the image of the spine. This roentgenogram is made by a synchronous shift of the tube and film during exposure, and the apparatus is so adjusted as to cause the rays to be focused at any desired depth. At this spot the rays are stationary, but objects above or below this depth are blurred by movements of the ray.

Slides are shown to illustrate the principle and results.

DISCUSSION

DR. RALPH A. FENTON, Portland, Ore.: May I ask Dr. Law whether, in his judgment, it is possible to get anything approximating the results of tomography by putting a piece of film down into the lower part of the pharynx in an envelope and making the roentgenographic image on that film covered with impermeable envelope? I should like some comment on that.

DR. CHEVALIER L. JACKSON, Philadelphia: I have long felt that the use of roentgen rays was of great help in the diagnosis of laryngeal disease, even in the days when one had only the lateral exposure. As Dr. Law has so well illustrated this morning, a great advance has been made in the development of tomography.

I have with me a slide which I thought might be of interest. It shows a neurofibroma in the larynx of a young woman. This is very well shown by the planigraph, while the conventional lateral view shows nothing but the obscuring of

the ventricle, that is, the loss of the ventricle shadow. In the planigraph the tumor is beautifully outlined, in contrast to the normal cord, the ventricle and the band on the other side. Dr. Van Loon, a member of the staff, removed this tumor by laryngofissure.

DR. HARRIS P. MOSHER, Marblehead, Mass.: A lateral plate should be taken in order to rule out any abnormality, such as the one Dr. Law showed. The old-fashioned plate is competent to show what one is going to deal with.

DR. O. V. BATSON, Philadelphia: I first learned about ossification of the larynx from Dr. Iglauer, and this is true ossification of the haversian system of the cricoid cartilage. While it can be called calcification, I think, if one is really precise, one should reserve the term for a pathologic process.

DR. SAMUEL IGLAUER, Cincinnati: Dr. Law stated that fluoroscopic examination of the larynx in the anteroposterior position was more or less futile. I should like to ask him whether it is not possible to see a limitation of motion of one cord in the anteroposterior view by using the fluoroscope. The Philadelphia school, I believe, describes the possibility of visualizing paralysis or impaired motion of one cord in the anteroposterior fluoroscopic position.

DR. FRANK R. SPENCER, Boulder, Colo.: In Omerod's last book, entitled "Tuberculosis of the Upper Respiratory Tract" (London, Bale Medical Publishers, Ltd., 1939), there are many laminagrams which are helpful in the diagnosis and differential diagnosis of tuberculosis. They are well made and bear out much of the information which Dr. Law has given for the diagnosis of laryngeal lesions.

In the past the roentgenogram has not been of great value in the diagnosis of tuberculous lesions of the upper respiratory tract because they can usually be seen by direct and indirect methods.

DR. CHARLES J. IMPERATORI, New York: Dr. Law points out that a lateral plate should be taken and that it should be compared with the tomograph. Certainly, the ventricles can be seen in the lateral plate, but in the tomograph they can be evaluated much better because there are two ventricles which are of exactly the same size. Also, the most important consideration of the tomograph is that one gets a good view of the subglottic area and is able to evaluate its condition. From a clinical standpoint that is of much importance because normally, even with the direct examination, unless the mass is large, it cannot be evaluated so well. I feel, therefore, that this method of procedure, in conjunction with the older method of the lateral exposure, is of great value.

My attention was originally directed to this by Dr. Law long before anything was known about the tomograph or the laminagraph. He had used this method in the location of foreign bodies during the last war, in France. Several years ago Dr. Felix E. Leborgne, of Montevideo, Uruguay, was in the United States and showed a number of these photographs and really revived interest in the method from the standpoint of the laryngologist, thus adding a new aid to diagnosis.

DR. FREDERICK M. LAW, New York: The method of viewing the larynx by means of a film inserted into the hypopharynx was employed many years ago. That was before the days of the planigraph, and it did give a very good view, but if any of you were present when the film was put into the hypopharynx, I don't think you would want to try it on yourself. I was enthusiastic when I first heard of it, and immediately tried it. When the planigraph came out and was first demonstrated so spectacularly by Dr. Felix E. Leborgne, of Montevideo, I had to build my own, as there was none on the market. It did not cost much but gives satisfactory results.

As to the fluoroscopic examination in the anteroposterior view, there may be times when one is able to visualize a cord. Probably Dr. Jackson could tell what Dr. Chamberlain thinks about this. The view with the planigraph is so satisfactory that one wastes one's time with a fluoroscopic examination, which is risky both to the examiner and to the patient.

Roentgen Treatment of Diseases of the Larynx. Dr. Maurice Lenz, New York.

Roentgen treatment has been directed to the larynx in an effort to control inflammation, papillomatosis and cancer. The usefulness of roentgen treatment of laryngeal tuberculosis is questionable. In cases of blastomycosis it is combined with iodides. For rhinoscleroma it is the treatment of choice. It is preferable to conservative surgery with multiple papillomas in adults, but has been replaced by estrogen therapy for papillomatosis in children. Roentgen therapy is more likely to fail with extensive, infiltrative, deeply infected types of cancer, especially if the growth invades cartilage. It competes successfully with laryngofissure in cases of small cancer limited to the anterior two thirds of one cord, and it is usually preferred to surgery in most cases of extrinsic cancer. It should follow laryngectomy in all cases in which microscopic examination of the removed specimen suggests that the operation has been incomplete. Of 89 patients treated with roentgen rays for cancer of the larynx at the Presbyterian Hospital from 1932 to 1936, 20 patients are now clinically free from cancer.

DISCUSSION

Dr. John H. Foster, Houston, Texas: I do not make any pretense of having deep knowledge of roentgen therapy. Whatever opinions I have formed have been those of a clinician. With my present experience I should subject any patient with undifferentiated cancer of the larynx to this treatment except when the cartilage was affected or when I thought roentgen therapy would cause breaking down of the cartilage. Therefore, the cartilage should be first removed and the larynx then treated with roentgen radiation. Of course, the cases representing the borderline between differentiated and undifferentiated carcinoma present the opportunity for a considerable difference of opinion.

I have been much interested for the last twelve years in roentgen therapy of papillomatosis of the larynx in children. I have yet to see such a condition in a child, who has not been subjected to operation, which would not clear up under roentgen therapy if I was able to get the roentgenologist to apply the dose I thought was indicated.

The question lies in the essential nature of the papillomas. In my opinion, they are not neoplasms in the ordinary sense of the word, but growths which result from some systemic condition.

I think the dose to be used for papillomatosis should be small, one that causes the papillomas to become absorbed. The failure to get the results I have obtained is due to the fact that roentgenologists will not use such a small dose, but insist on one that will destroy the growths. In my town I have only one roentgenologist whom I can depend on to use the dose I advocate.

In the first case I reported in 1930 the patient came back to me in ten years with a single papilloma of the vocal cord, which I removed. Another patient returned with one large papilloma at the site of a tracheotomy wound, and neither of these has had a recurrence of the general papillomatosis which was present at the time of my first general treatment. The patients have perfectly normal larynxes after the papillomas are absorbed, and one cannot tell that there has ever been anything the matter.

Dr. John F. Barnhill, Miami, Fla.: Dr. Lenz has touched on a point which has interested me for many years; that is: Why is a malignant growth of the arytenoid cartilage so frequently fatal and one of the cord itself not so fatal? My final conclusion is that it is entirely a question of the lymphatics.

Doubtless some have seen an illustration, appearing in one or two books, which shows the distribution of the capillary lymphatic trunks and their union inside the larynx. It will be remembered that there is scarcely any lymph channel on the cord itself. Many investigators say that there are none on the cords, but if one studies the arytenoid cartilages and the region above and below them, in other words, the region where extrinsic cancer develops, one will find the densest distribution of lymph vessels; so I think it is a question of lymph distribution.

DR. MERVIN C. MYERSON, New York: Dr. Lenz has given a conservative, lucid, praiseworthy and modest presentation. I should like to touch on several unrelated points.

In the matter of tuberculosis of the larynx, I doubt whether American laryngologists will ever subject their patients to roentgen therapy. This is because it is becoming more universally understood that only in the larynx which has the ability to produce fibrous tissue can one expect healing. Therefore, any form of therapy applied to tuberculosis will give results in cases of the fibrous, or productive, type of the disease in the larynx. Nothing will help the ulcerating, disintegrating type of lesion. It is safe to say that the chronic, productive type of tuberculosis of the larynx will heal without any laryngeal treatment. It may heal a little faster if one applies the cautery and certain stimulating medicaments.

In the matter of carcinoma of the larynx, the chief consideration is the cartilages and the injury to which they are subjected by roentgen therapy. There seems to be considerable difference of opinion among laryngologists who are interested in surgery of the cancerous larynx as to whether a patient who has had irradiation should be operated on. Many surgeons will not operate on patients who have had roentgen therapy. I have come to believe that such patients, with telangiectatic and stiff subcutaneous tissue, can be operated on with some expectation of good results. I should like to ask Dr. Lenz whether it is possible for the roentgen therapeutist to carry out a technic which would leave an unexposed area in the midline of the neck, so that if future operation is necessary or decided on the vitality of the tissues through which the surgeon cuts will not be too greatly compromised.

I should also like to pose the question: Are we not, as laryngeal surgeons, cleaving too closely to the indications for surgery of the larynx that were laid down thirty or more years ago? Might it not now be proper to revise our attitude on the management of cancer of the larynx that is not entirely intrinsic?

Last week, at a meeting of a local medical society, I reported 2 cases in which I operated, but in which ordinarily surgery would not have been resorted to and the response to irradiation would certainly not have been favorable. In each case the growth was an undifferentiated squamous cell carcinoma.

The first case was that of a man who had an enlarged mass of glands on the left side of the neck and bilateral superglottic extension of the cancer. I removed the gland and a week later the larynx. The man survived for three and one-half years and died of a metastatic lesion in the lung. I think that if one can give a man a comfortable existence for three and one-half years, operation is well advised.

In the other case, a cancer must have originated in the epiglottis and extended to the larynx. It covered the larynx so that nothing could be seen but an enormous fungating mass. It had also extended into the base of the tongue. I removed this mass through a pharyngotomy opening. The man is still alive, thirteen or fourteen months after the operation. He received a full course of roentgen therapy, and the skin was not too badly compromised when he came back for operation. I removed the larynx last week and encountered an abscess which I did not anticipate. I hope his tissues will heal.

DR. HENRY B. ORTON, Newark, N. J.: About four or five years ago, at a meeting of the Academy of Medicine in New York, when I was on the program with Dr. Lenz, I stated that I had yet to see a case in which carcinoma of the larynx was cured by irradiation. Apparently, at that time I was right, but I now make public the statement that I have seen cases in which Dr. Lenz cured the lesion. He has given one of the most honest reports of roentgen therapy of the larynx that I have heard. I shall confine all my remarks to carcinoma of the larynx.

I operate on the larynx whenever surgical treatment is possible. In cases of intrinsic cancer, in which Dr. Lenz wishes to use roentgen therapy more often in the type in which the growth is at the junction of the anterior and the middle third of the cord, I still think surgical intervention is the method of choice.

In cases of the so-called extrinsic type, involving the epiglottis, the aryepiglottic folds and the arytenoid cartilages, if the patient is in good physical condition, if there are not too many affected glands and if they are freely movable, I still prefer to do a radical operation and supplement it with roentgen therapy.

Dr. Lenz stated that in 1 of his cases a carcinoma was cut across. I should like to ask him if it was high up in the epiglottic region or at the level of the anterior commissure, for that might bring into question the space of Boyer. In the case of a carcinoma penetrating the anterior commissure, one will cut through it, and for that reason in every laryngectomy which I do the hyoid bone is removed with the larynx.

Dr. Frederick A. Figi, Rochester, Minn.: This is the frankest statement on the subject that I have ever heard from a roentgenologist. The experience of my associates and myself in the roentgenologic treatment of blastomycosis and rhinoscleroma corresponds with that of Dr. Lenz. With regard to the treatment of papillomas, we use irradiation somewhat, but we are not as convinced as to its efficacy as Dr. Foster is. As a matter of fact, I have seen several cases of papillomas in children in which malignant changes developed after irradiation, even at the age of 5 or 6 years. Whether these changes would have occurred had not irradiation therapy been given is of course open to question, but it was our belief that the irradiation was a factor in the development of the malignant change.

It is still our feeling that any laryngeal carcinoma which is operable should be dealt with surgically. One can do much more radical removal through a laryngofissure than some of the older writers on the subject would have one believe. As a matter of fact, some patients, even with extensive carcinoma of the larynx, who are subjected to laryngofissure do remarkably well.

Dr. Jackson may recall a patient on whom I happened to be operating when he visited us five or six years ago—a man with a fixed carcinoma of one side of the larynx, a grade 2 lesion. I had advised laryngectomy and was firmly convinced even after exploring the growth through a tube, that such an operation should be performed. However, the patient would not tolerate a laryngectomy; so we electrocoagulated the growth thoroughly, removing the involved cartilage. The patient is still perfectly well.

Dr. Myerson's question whether a patient whose laryngeal carcinoma has been irradiated should be later subjected to radical surgical treatment is of interest. In many such patients healing takes place almost as well as in a patient who has not had intensive irradiation, and we have seen a number who remained well for some years.

Radiation therapy for carcinoma of the larynx cannot be dismissed too lightly in favor of surgical treatment. One cannot help a certain amount of enthusiasm when one sees patients with frankly inoperable cancer of the larynx, with metastatic nodes in the neck, who after fractional roentgen irradiation are without evidence of recurrence for several years. How much longer they will remain free from a malignant process is, of course, questionable. But a number of these patients obviously had only a few months to live at the time they were referred for radiation therapy.

With regard to the reason that lesions involving the arytenoid cartilages are less responsive to irradiation than those involving the epiglottis, I wonder whether it is not a difference in the activity of the growth. Ordinarily, lesions originating on the epiglottis are of a low degree of malignancy and metastasize late, while those originating on the arytenoid cartilage are, with few exceptions, highly malignant and metastasize actively.

Dr. John D. Kernan, New York: I have seen Dr. Lenz's work almost since its inception, and I have been struck by the great improvement which has occurred in the application of roentgen therapy of diseases of the larynx. I can remember when the cancer was cured by roentgen rays and the whole larynx would slough out. I recall 1 or 2 cases of that kind.

Dr. Lenz did not mention several patients whom he has recently treated for me because they do not represent five year cures, but they have been well for two or three years. Not only did the carcinoma disappear, but it left no trace, either inside or outside the larynx. There is no telangiectasis or scar tissue in the skin. The patients have perfect vocal cords with a perfect voice; so early carcinoma of the larynx, when it is limited to the cord and the cord is mobile, is cured as Dr. Lenz treats it.

Laryngectomy is not a cure for carcinoma of the larynx. It is analogous to amputation of the foot caught in the trap, but to have a carcinoma disappear without trace is a cure. So it may actually be said that Dr. Lenz cures cancer of the larynx.

Dr. Samuel Salinger, Chicago: If Dr. Lenz reflects the generally accepted views among roentgen therapeutists on this point, I should say that a great change has taken place in their attitude, for he admits that the histologic appearance of the tumor is no longer any criterion for the employment of roentgen therapy. He admits that infiltration and fixation are contraindications to roentgen therapy, and he has shown his best results with the type for which surgery has been so eminently successful, namely, the isolated lesion with the movable cord. The question, then, arises whether in choosing roentgen therapy instead of surgery, with its known background and record, one is willing to subject the patient to six or seven weeks of discomfort and pain in order to achieve the results which Dr. Lenz has shown.

I still feel that when there is a difference of opinion or when there is disinclination on the part of the patient to subject himself to operation and he submits to irradiation, there must be a time during the course of the radiation therapy when it should either be interrupted or be completed—interrupted for the purpose of resorting to surgery, for I still feel that after a complete course of irradiation there is some risk in operation.

Dr. Lenz admits that the margin of safety between the point at which the tissues have received their maximum safe dose and the point at which damage can be done is small. In cases in which the patient insists on irradiation, it has been my practice to interrupt the irradiation and insist on operation if I determine that no further progress is being made by the irradiation.

My experience with postoperative irradiation has been good, and I agree with Dr. Lenz that it can be given with safety and that the results are good.

Dr. T. E. Walsh, St. Louis: What is the total dose Dr. Lenz employs in cases in which irradiation follows laryngectomy?

I have seen recently a case in which laryngectomy was done about a year ago and was followed by radiation therapy. The patient has a recurrence in the pharyngostomy opening, and I wonder how much more therapy can be given safely, whether it can be given at all after his original irradiation and whether a time factor should be allowed between the first dose of radiation and any subsequent dose.

Dr. Thomas C. Galloway, Evanston, Ill.: In Coutard's report on his method, which is probably the best in an extensive series, 27 per cent of the patients were stated to be free from disease for five years. Subsequently, however, about one third of these patients had a recurrence.

I should like to ask Dr. Lenz what limits he would fix for the time after which one may assume a definite cure after roentgen therapy.

Dr. Charles J. Imperatori, New York: I wish to report briefly on 2 of Dr. Lenz's cases with which I had something to do, the case of the rhinoscleroma and the case in which I did the laryngectomy after the radiation therapy. The patients remained well.

Has Dr. Lenz come to any conclusions about the indications for radiation therapy? In which cases should radiation and in which should surgical treatment be given? I do not mean cases of the advanced stage in which a laryngectomy is required or cases in which the tumor is evidently beginning to invade a little

beyond the area of the growth on the cord. I am speaking about the early stages, and I feel that the proper treatment in cases of this type is surgical.

Has Dr. Lenz had any experience with the role that water plays in irradiation of the tissues? Recently Dr. Jack De Ment has published a book entitled "Fluorescent Chemicals and Their Application" (New York, Chemical Publishing Company, 1942). There is a reaction which takes place in certain fluorescent materials when they are injected into the tissues and the tissues are irradiated. The ultraviolet rays apparently are stimulated in such a way that nuclei are destroyed. The work seems to offer an entirely new principle of therapeutic approach, and I should think that Dr. Lenz might be able to say whether or not this procedure is a proper therapeutic method and one which might help.

The use of "heavy" water has been suggested in some of these cases, purely from an experimental standpoint. Whether or not there have been any cures with the injection of "heavy" water into the tissues followed by irradiation I do not know.

Dr. Maurice Lenz, New York: Dr. Foster has brought out his experience with low voltage roentgen therapy, and he has published a number of articles on it which prove his point. However, I think the answer to therapy of papillomas in children was indicated by Dr. Broyles. Estrogen therapy is probably simpler and better. However, as a roentgenologist, I see no reason why roentgen therapy should not be used if the laryngologists insist on this form of treatment.

Dr. Barnhill made a point about the lymphatics about the arytenoid cartilage. I wonder how one can correlate the extension to the lymph nodes with this observation. The patients die with severe local infection. Many of them have a necrotic wound, and it seems to me that the necrosis of the cartilage, along with the long-standing chronic infection, is sufficient to explain death. But there is no question about the point, which some one else brought out, that in the cases of arytenoid involvement there are many more metastases.

In discussing the lack of indication for roentgen therapy, Dr. Myerson brought out the natural tendency of laryngeal tuberculosis to heal. In general I agree with him, but Rickmann's work cannot be lightly dismissed (Bacmeister, A., and Rickmann, L.: Die Röntgenbehandlung der Lungen- und Kehlkopf-Tuberculose, Leipzig, Georg Thieme, 1924). Rickmann was in charge of a sanatorium and was not a radiologist. He reported on two large series of patients, one of which had all the sanatorium care plus roentgen therapy and the other the sanatorium treatment without the roentgen therapy. He claimed that the results in the first series were better. As I said, I do not believe that roentgen therapy should be carried out for tuberculosis of the larynx except in a sanatorium or hospital for tuberculous patients.

Dr. Myerson also asked the question: "Can irradiation be given so as to leave a midline space where it would be safe to operate?" Ordinarily this is done, because one is afraid of overlapping action and getting a burn in the midline; so I think it is safe to operate in cases of cancer of the larynx after irradiation.

Again, I have no quarrel with him, Dr. Orton or any one else who has successfully operated on extrinsic cancer. Every once in a while I am sure it can be done, but I wonder of, say, 100 patients with this type, how many remain well for ten years. That is the only criterion as to whether one should operate or irradiate.

It is unfair to attribute every instance of necrosis of the cartilage following roentgen therapy to such treatment unless one ean show microscopically that the cartilage was not invaded by cancer cells.

Now I come to a difficult question: Which of the patients with early cancer should be subjected to surgical and which to roentgen therapy? I do not know. I have not enough experience. I can only say that of the few patients I have treated, the majority have remained well. I think this is a question for individual decision. My usual procedure is to tell the patient: "If you have a laryngofissure, you are going to be through with all your treatment in a week. If you have roentgen therapy, it is going to take at least a month, and you will have several

weeks of discomfort. The voice in the end will be better." I think that is fair. Then the patient decides. Recently, one of the surgeons at the Presbyterian Hospital and I saw a case jointly and put the decision up to the patient. The surgeon pressed his point a little stronger and so operation was performed. In this case there was a peculiar complication in that severe subcutaneous emphysema of most of the body developed. None of us, the surgeon, the patient or I, expected it.

Dr. Imperatori brought up the point about heavy water. All types of heavy elements have been tried in combination with roentgen therapy. The difficulty with all of these methods is this: The elements do not select the cancer cell exclusively but deposit just as heavily in the normal cells; therefore there is no advantage in this form of treatment. For instance, when colloidal lead was used to treat cancer, just as much of the lead was deposited in the bone marrow as in the cancer with the result that the patients all died of anemia, in spite of numerous transfusions. Local injections of heavy water within the area of the larynx will deposit just as much in the normal cells as in the cancer cells, and therefore it does not seem an advantageous form of treatment.

I have known of Dr. Orton's views, and no one respects his beautiful surgical work more than I do; it is a revelation to me to have him say that he is satisfied that I do cure cancer in some cases.

Again, I have no quarrel with any one who wants to treat the early stages by laryngofissure and cordectomy, as Dr. Orton or Dr. Figi would do. I offer the suggestion that, if for any reason the laryngologist or the patient decides against operation, roentgen therapy gives him a chance of cure of an early lesion.

Roentgen exposure of cells produces maturation; that is, the cells become more mature and more differentiated; therefore I doubt whether the case which Dr. Figi cited in which a carcinoma developed after roentgen therapy in a child could be attributed to the irradiation, but then, again, that is a moot point.

Occasionally I do as Dr. Salinger suggests. When, after a few weeks' treatment there is no regression of the tumor, I ask the laryngologist to operate. I have done that on several occasions.

Dr. Walsh wanted to know whether a patient who has had a full course of treatments can get another treatment. My experience has been this: When I administer, with 200,000 volts and a 1 to 2 mm. filter of copper, a daily dose of not more than 125 to 150 r to each of the laryngeal fields, and a total dose of about 3,000 r to each of the laryngeal fields, provided that the fields are not larger than 6 by 8 cm., I have repeated that dose two and three years later.

In 1 case I repeated a dose in six or seven months. The cancer was not cured; but the tissues remained healthy.

I might say here, also, that in cases of recurrence in the tracheotomy wound which one occasionally sees after laryngectomy I have not been successful in arresting the disease.

Dr. Orton asked where the extension was in those cases in which the cancer had been cut across. I remember that in 2 of them there was penetration through the thyroid cartilage.

Dr. Galloway asked when cancer may be considered cured after irradiation. When is cancer cured after laryngectomy? I think the answer is the same to both questions. I don't know when it is cured. I should say that an arbitrary figure of five years, such as the surgeons use, is fair.

Chondroma of the Larynx. Dr. E. Trible Gatewood, Richmond, Va.

This paper was published in the August 1942 issue of the Archives, page 187.

DISCUSSION

Dr. E. R. King, Cincinnati: Dr. Gatewood referred to a case of a young man in which sudden dyspnea developed. Was the tumor a chondroma?

Dr. E. Trible Gatewood, Richmond, Va.: Dr. Jackson had a case like that six or eight years ago which was not reported. The tumor was tracheal because

the site was in the region of the first ring of the trachea rather than in the cricoid region.

Anterior Commissure Tendon. Dr. Edwin N. Broyles, Baltimore.

Serial cross sections of the region of the anterior commissure of a normal larynx demonstrate the firm attachment of the vocal cords into the midline of the thyroid cartilage by means of the anterior commissure tendon. The internal perichondrium is not firmly attached laterally, but in the midline the tendon blends into cartilage, with no plane of cleavage.

In the lower portion of the thyroid cartilage there is no firm attachment, the tendon sweeping around and not entering the cartilage.

It is suggested that this crista of the thyroid cartilage be removed along with the diseased cord in cases of carcinoma in the region of the anterior commissure.

DISCUSSION

Dr. Henry B. Orton, Newark, N. J.: I was much interested in seeing this demonstration, Dr. Broyles, because it is what I have been talking about for a number of years. If any growth involves that area, I should perform a total laryngectomy. It is from the area of which Dr. Broyles speaks that a growth penetrates the cartilage and enters the space of Boyer.

Dr. Chevalier L. Jackson, Philadelphia: I found this presentation of Dr. Broyles most instructive and illuminating, and it has certainly inspired me to have even more respect for the lesions at the anterior commissure than I had before. But I do not think that it absolutely disqualifies the anterior commissure type of laryngofissure, in which one goes through the cartilage from the outside and then separates the lesion with the adjacent tissue on each side before making the excision of the tumor. I think statistics prove that this operation has justified itself in the cases of lesions of the anterior commissure.

It is true that statistics show that laryngofissure in these cases is not attended with as high a percentage of cures as when a lesion involves the middle third of the cord. However, I still believe there are cases of small lesions of the anterior commissure in which laryngofissure is justified.

Dr. Edwin N. Broyles, Baltimore: The chief point I wanted to bring out was the fact—and I know of Dr. Jackson's success with the anterior commissure operation—that the perichondrium does not extend completely around the angle of the thyroid, and when one cuts through the cartilage anteriorly one must make a careful examination of the specimen, especially of the anterior portion, to be sure a little carcinoma has not been left in that region.

CASE REPORTS

Ganglion Cell Neuroma of the Pharynx. Dr. John H. Foster, Houston, Texas.

This paper was published in the September issue of the Archives, page 372.

DISCUSSION

Dr. John F. Barnhill, Miami, Fla.: What nerve was supposed to be involved?

Dr. J. H. Foster, Houston, Texas: There was no direct connection with any nerve that I know of.

Dr. John F. Barnhill, Miami, Fla.: Could you have a neuroma without its having connection with a nerve?

Dr. John H. Foster, Houston, Texas: I suppose the nerve tissue there had some connection with the sympathetic nervous system because it was close to the superior cervical sympathetic ganglion.

Rhinoscleroma Involving the Upper and Lower Respiratory Tract. Dr. F. W. Dixon, Cleveland.

This article will be published in full in a later issue of the Archives.

DISCUSSION

Dr. Louis H. Clerf, Philadelphia: I had the opportunity of seeing this patient. I have also seen 2 others, who have no nasal lesions. The question of therapy has been of interest because 1 of these patients has extensive laryngeal involvement. Tracheotomy has been done and radiation given in small doses at frequent intervals, with a certain amount of relief. If the therapy is discontinued, the lesion flares up rather promptly. In the other patient the condition is quiescent and he is getting on well. I am interested to know about the status of radiation therapy for this lesion.

Vincent's Ulceration of the Soft Palate. Dr. K. M. Houser, Philadelphia.

This paper was published in full in the August 1942 issue of the Archives, page 236.

DISCUSSION

Dr. Thomas C. Galloway, Evanston, Ill.: What treatment did Dr. Houser use for this infection?

Dr. Henry M. Goodyear, Cincinnati: I should like to mention a preparation which I do not believe has been used often in the treatment of Vincent's angina, and that is a 10 per cent solution of copper sulfate.

In many cases in which the arsenical preparations and the various drugs commonly recommended for Vincent's angina have been used without success the condition clears up rapidly with 10 per cent copper sulfate. In fact, I have seen gingivitis which had not responded when treated by dentists for weeks clear up in a week or ten days when the gingiva were swabbed once a day with a 5 or 10 per cent solution of copper sulfate.

Dr. T. E. Carmody, Denver: For a number of years I have been using copper sulfate, 10 to 15 per cent in water, and then I give the patient a 25 per cent solution in glycerin to use himself. It works almost perfectly. It is the best treatment for Vincent's angina, either around the gums or in the tonsils, that I have seen.

Dr. K. M. Houser, Philadelphia: I think all the standard treatments, with the exception of copper sulfate, were employed. They included sodium perborate and hydrogen peroxide and irrigations of the crypts of the tonsils themselves. Incidentally, in this case the tonsils appeared perfectly normal until they were pressed on. It was not at all the typical tonsil of Vincent's angina. As a matter of fact, as this case was unusual, I am not sure whether or not Vincent's organisms caused the condition. I am reasoning backward, but the sore cleared up with the use of mercuric cyanide. I also used silver nitrate.

I cannot give the figures on the differential cell count, but I should say it was not in any way suggestive of infectious mononucleosis. There was some leukocytosis. This persisted over a long period.

Abscess of the Parapharyngeal Space; Massive Hemorrhage; Recovery. Dr. W. E. Grove, Milwaukee.

This case concerns a boy 11 years of age whose tonsils and adenoids had been previously removed and in whom there developed successively a cold in the head, stiff neck, swollen cervical glands, otitis media and a swelling of the lateral pharyngeal wall posterior to the posterior pillar. An abscess in the left parapharyngeal space was opened externally, at which time the internal jugular vein was ligated. Six days later a hemorrhage occurred, which was controlled by a tight pack in the external wound. Two days thereafter there was a second alarming

hemorrhage, of from 300 to 400 cc. of blood, from the external wound. The bleeding vessel was identified as the ascending pharyngeal artery, which was ligated. The bleeding ceased, and the patient recovered after a stormy convalescence, which included a mastoid operation.

DISCUSSION

DR. HENRY ORTON, Newark, N. J.: I had 2 cases in which massive hemorrhage followed a parapharyngeal abscess. It is not a pleasant thing to see.

Amyloid Tumor of the Larynx. DR. LOUIS H. CLERF, Philadelphia.

This paper was published in the September 1942 issue of the ARCHIVES, page 377.

DISCUSSION

DR. ERNEST M. SEYDELL, Wichita, Kan.: I wish to report a similar case—that of a man about 62, in whom the growth was subglottic. It was not removed. He had radiation treatment and lived for a number of years, dying of pneumonia.

DR. FREDERICK A. FIGI, Rochester, Minn.: Dr. Clerf's case, in my experience, is unusual in that the involvement was unilateral at the time the patient came under his care. More frequently, in my experience the involvement has been bilateral, and commonly there has been diffuse infiltration of both vocal cords, and often of the ventricular bands as well, in some instances extending pretty well down into the upper portion of the trachea.

In a case I had recently, that of a man aged 62, there was pronounced obstruction from the tumor at the time he came under my observation. He gave a history of progressive hoarseness and respiratory difficulty, of approximately three years' duration. Examination showed diffuse infiltration involving both sides of the larynx, chiefly in the subglottic region, where a sizable mass was attached to the inferior aspect of each cord, there being only a narrow chink between these masses. The bronchoscopist was unable to insert even a small instrument. An emergency tracheotomy had to be done during the course of the examination because of his dyspnea. About two weeks later I explored the tumor through a laryngofissure and found a mass measuring about 1.5 by 2.5 cm. along the mesial and inferior aspects of both cords. This was excised, and below this, extending to the level of the fourth tracheal ring, there was diffuse infiltration of the subglottic portion of the larynx and the tracheal wall. That portion of the tumor I removed with sharp curets until a lumen of fair or almost normal size was established.

A skin graft was then applied to reline the larynx. I removed the soft rubber dilator about twelve days later. At this time the tracheotomy could be corked, and a few days after that I removed the tube. The man's voice was rapidly becoming normal, and he was breathing without any difficulty.

DR. CHEVALIER L. JACKSON, Philadelphia: I should like briefly to mention a case in which, as in Dr. Figi's, the tumor was bilateral and rather diffuse. The patient, a schoolteacher about 30 years of age, had had serious impairment of her voice for about two years before I first saw her. The first diagnosis I made was phonation with the ventricular bands, and I made that diagnosis without having any explanation for the development of the condition. But when I saw the patient the next time, about two years later, I decided to make a direct laryngoscopic examination and take a little tissue for biopsy. There had not been much change in the tissue, but I have slides which established the diagnosis of amyloidosis of the larynx. The patient has gone on now for another three or four years, without any change in her symptoms. The growth seemed too diffuse for any attempt at removal. She did not have dyspnea; so tracheotomy was not necessary.

I should like to know what treatment Dr. Clerf would advise in a case like this, in which the lesion was not localized, and not obstructive, and in which there was certainly no prospect of improving the voice by radical excision.

DR. LOUIS H. CLERF, Philadelphia: I am afraid I cannot answer Dr. Jackson's question.

I do not know why amyloid disease develops, with isolated or more or less localized deposits. I suppose it is a matter of waiting until the patient gets worse and then doing what Dr. Figi did.

DR. FREDERICK A. FIGI, Rochester Minn.: Why not try irradiation?

DR. LOUIS H. CLERF, Philadelphia: I have had no experience with it.

DR. FREDERICK A. FIGI, Rochester, Minn.: I think that used to be our routine treatment; then excision and skin grafting.

Osteitis Fibrosa Cystica of the Left Frontal Sinus. DR. JOHN C. KERNAN, New York.

This paper was published in the September 1942 issue of the ARCHIVES, page 385.

DISCUSSION

DR. THOMAS C. GALLOWAY, Evanston, Ill.: I should like to ask whether there was any evidence of parathyroid disease.

DR. JOHN C. KERNAN, New York: There was none.

Granuloma of the Nose. DR. D. M. LIERLE, Iowa City.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. N. CANFIELD, New Haven, Conn.: I had a similar experience during the past year, and in many respects this case is identical with one I have had under observation in New Haven. The patient was about 10 years older than the woman whose case Dr. Lierle reported and had had long-standing, chronic suppuration in the maxillary sinus preceding the appearance of the granuloma. I was somewhat more energetic in my surgical removal of the sloughing and necrotic tissue, because I thought if I carried it out thoroughly, I might be able to place zinc peroxide in contact with the organism.

The only offending organism which I could find in my case, besides the secondary invaders, was the hemolytic streptococcus. Complete sloughing of the upper lip and of the left side of the face took place, and inanition, secondary anemia and terminal pneumonia developed.

As in Dr. Lierle's case, I tried every possible method to stop the infection, entirely without success. I thought at one time that zince peroxide was causing some recession of the process, as Dr. Lierle did in his case, but it became evident that the drug had no effect.

I have no further suggestions to make about the condition except possibly the use of an agent known as penicillin. I cannot tell you whether that drug is available, but it is now being worked on in the laboratory of the National Research Council.

DR. MERVIN C. MYERSON, New York: This unusual case recalls to my mind a tropical disease known as gangosa. Some fifteen or sixteen years ago I had a case of gangosa in an American who had never been out of this country. It is entirely possible that Dr. Lierle was dealing with gangosa in his case; I wonder whether he thought of that possibility. Gangosa has no specific features, but clinically it acts exactly like the disease in Dr. Lierle's case.

DR. ERNEST M. SEYDELL, Wichita, Kan.: I saw a similar case in consultation three or four years ago in which the patient was a man about 65 years of age. At the time I saw him his nose was entirely destroyed. The upper lip was not involved. The man lived about four or five months after I saw him and then died of a severe hemorrhage. In this case everything was done that could be thought of, without any effect.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: I had a case in which the condition started in a similar fashion. I do not know how far it would have progressed, but I used energetic coagulating diathermy. I think that is the treatment of choice for such a lesion, at least when it first appears.

This condition is a type of anaerobic infection which becomes indurated, and the induration inhibits the effects of the zinc peroxide. I wonder what kind of zinc peroxide Dr. Lierle used, and whether it was properly activated. In such a lesion one must destroy every pocket beyond the limit of the infection, and then zinc peroxide can be employed prophylactically to considerable advantage.

DR. D. M. LIERLE, Iowa City: The zinc peroxide was properly activated. The fault lay in my inability to get into the deeper tissues early. I did not think of the disease gangosa, which Dr. Myerson spoke about; is there anything to be done for it?

Vitamin Deficiency with Laryngeal Symptoms: Report of a Case. DR. R L. GOODALE, Boston.

A woman aged 66 was first seen on Sept. 12, 1941, complaining of hoarseness and dysphagia of three months' duration. In addition, she had had several attacks of soreness of the tongue and lips, with puffiness around the mouth at times. There was a history of improper diet, due to loss of teeth.

Examination showed swelling involving the arytenoid cartilage, false cord and aryepiglottic fold on the left side, with complete loss of motion of the true cord, which was otherwise normal.

Examination of the esophagus showed loss of peristalsis, barium sulfate remaining in the esophagus when the patient was in the horizontal position.

Biopsy of tissue from the laryngeal swelling did not reveal a malignant growth. The Hinton and cytologic reactions of the blood were negative. Except for loss of motion in the left vocal cord, the neurologic examination showed nothing of importance. The patient was given large doses of a preparation of vitamin B complex, with immediate improvement; the soreness of the tongue and lips and the swelling in the larynx rapidly disappearing, and motion of the left vocal cord returned to normal within a few weeks. There has been no recurrence. The patient was last seen March 16, 1942, when she had completely recovered.

Septic Thrombophlebitis of the Neck: Report of a Case with Serious Complications. DR. SAMUEL IGLAUER, Cincinnati.

This paper was published in the September issue of the ARCHIVES, page 381.

DISCUSSION

DR. F. W. DIXON, Cleveland: In 1937, when I was visiting the clinic of Dr. Claus of Berlin, I observed his use of a wide and free incision. The amount of thrombosis revealed was not always extensive. After finishing his dissection of the neck, he immediately removed the tonsil.

DR. T. E. CARMODY, Denver: In what position did Dr. Iglauer place the patient? Did he operate in the Trendelenburg position, or did he elevate the foot of the bed to keep the infection from traveling downward?

DR. SAMUEL IGLAUER, Cincinnati: With regard to what Dr. Dixon said, Claus, according to his last paper, has changed his procedure a bit, inasmuch as he does not always remove the tonsil after operating on the neck, chiefly because the patient is very ill and if one takes out the tonsil, one interferes with his swallowing and nutrition.

In this particular case, however, it would have been advisable to remove the tonsil right at the operation; I intended doing that, but was somewhat delayed by some bleeding in the neck; so I decided to postpone it.

As to Dr. Carmody's question, the involvement of this patient's lung was undoubtedly through the blood stream; so I do not see that posture would have

had any effect. I think the patient was admitted with emboli in the lung. The first, or at least one, provisional diagnosis was pneumonia. She had undergone thoracotomy in the surgical service.

DR. JOHN F. BARNHILL, Miami, Fla.: Where did the original infection come from, the tonsil?

DR. SAMUEL IGLAUER, Cincinnati: From the peritonsillar region.

DR. JOHN F. BARNHILL, Miami, Fla.: Did she have quinsy to begin with?

DR. SAMUEL IGLAUER, Cincinnati: Apparently, because as the tonsil was being removed, the pus came out from the peritonsillar or pharyngomaxillary region, since the neck was already open. The exploration in the neck showed no pus.

DR. JOHN F. BARNHILL, Miami, Fla.: What is the relation of the quinsy to a pharyngomaxillary abscess? Did the patient have a pharyngomaxillary abscess during the progress of this condition?

DR. SAMUEL IGLAUER, Cincinnati: I think quinsy is usually the source of a parapharyngeal or pharyngomaxillary abscess.

Histoplasmosis of the Larynx. DR. L. W. DEAN JR., St. Louis (by invitation).

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Résumé of Case Reports Presented Since 1939: Results and Present Status. DR. CHARLES J. IMPERATORI, New York.

The first case is that of a patient presented in 1939 and again in 1940 at the annual meetings of the American Laryngological Association held at Rye, N. Y. The details are found in the *Transactions of the American Laryngological Association* (**61**:257, 1939; **62**:47, 1940). This young man, now 22, has stenosis of the larynx. He appears to be in perfect health, is 6 feet (182 cm.) in height and weighs 185 pounds (83.9 Kg.) and works as a machine operator thirty-six hours a week. The stenosis resulted from radiation therapy and a poorly executed radical operation performed in the attempt to relieve papillomatosis of the larynx. His larynx has been kept open by core molds. Previously there was total laryngeal stenosis. The two laryngostomies left an opening in the neck of approximately 2½ inches (6 cm.). This opening was closed to one of approximately ½ inch (1 cm.) through which most of his breathing is done, the laryngeal opening being the size of a 16 French catheter.

When the case was previously presented, the question was asked whether a plastic operation might be performed, possibly a bridge implantation of cartilage anteriorly. In the discussion that took place this procedure was not advised, mainly because of the great possibility of failure, with the establishment of a worse condition. Incidentally, this young man has a good pharyngeal voice. There is no movement whatever in the tissues of his larynx.

The second case is that of the little lad B. S., who was presented twice before the association and whose condition was a congenital tracheoesophageal fistula that was closed by a plastic procedure transtracheally (*Tr. Am. Laryng. A.* **61**:147, 1939; **62**:148, 1940). The boy's condition can be reported as cured. He is now 10 years old, weighs 60 pounds (27.2 Kg.) and has grown approximately 6 inches (2.7 cm.) since the last time he was presented. (In 1940 he weighed 40 pounds [18.1 Kg.].) He takes part in all school activities and ranks well in his classes, both in regard to behavior and to studies.

This case was most interesting because of a physician's attempt to produce a dilation for relief of cardiospasm. The child was given a long string to swallow, with the idea of passing the instrument down the string as a guide. As pointed out by several members of this association and by others, particularly Dr. Porter P. Vinson, of Richmond, Va., a string should never be used when there is a possibility of tracheoesophageal fistula, because of the danger of the string's

winding itself around the tissues, between the normal laryngeal opening and the fistulous opening. Such an accident occurred in this child, and it was with difficulty that the foreign body, which amounted to 10 yards 26 inches (9.8 meters) was removed by my former associate, Dr. David Jones, at the Manhattan Eye, Ear and Throat Hospital. However, at the time the string was swallowed the diagnosis of tracheoesophageal fistula had not been established.

Two operations on the fistula, approximately two months apart, resulted in permanent closure.

It is with pride that I call attention to the cure in this case.

With regard to a 7 month old child with pronounced dypsnea due to compression of the structures of the neck by hygroma cysticum coli (*Tr. Am. Laryng. A.* **62**:51, 1940), the attending physician reports that the child is well and growing normally.

It would seem that the treatment, consisting of aspiration and injection with a mild sclerosing fluid, is deserving of a trial in cases of the condition, before attempting surgical intervention.

With regard to the 72 year old patient with the lymphoma of the larynx (*Tr. Am. Laryng. A.* **63**:134, 1941), he has gained 6 pounds (2.7 Kg.), his weight now being 224 pounds (101.6 Kg.), and for a patient of his years he looks exceptionally well. There has been no recurrence of the growth, and his voice is entirely normal. This patient was operated on in October 1940, and about a year afterward there occurred enlargement of a lymph node at the angle of the jaw. This rapidly increased in size, so that it was approximately 1½ inches (3 cm.) in diameter. Carious teeth with apical abscesses were removed, and there was prompt absorption of the intumescence. Recent examination showed no further enlargement of any of the lymph nodes, and the patient reports that he is in splendid condition.

The reasons for reporting this case are the size of the growth, the method of removal (laryngofissure) and the result with respect to the voice, which is normal, and the present condition.

DISCUSSION

DR. THOMAS E. CARMODY, Denver: I should like to ask Dr. Imperatori to explain how he closed the fistula between the trachea and the esophagus, for it was very cleverly done.

DR. FREDERICK A. FIGI, Rochester, Minn.: I should like to ask Dr. Imperatori why he has not closed the stenosed larynx in the first case.

DR. SAMUEL IGLAUER, Cincinnati: Was the child with the tracheoesophageal fissure ever given barium sulfate or iodized poppyseed oil to swallow?

DR. CHARLES J. IMPERATORI, New York: First, as to how the fistula was closed, an external incision was made into the trachea, which was opened and retracted. The fistula, a slit in the tracheal wall, in the space in which there were no rings, could be seen, and with a probe one could enter the esophagus.

The mucous membrane was separated, and an incision was made around the fistula. Because it required two procedures, I felt at that time that if a purse string suture was used, puckering of the posterior tracheal wall, and perhaps some difficulty of breathing, would result. The expansibility of the trachea on coughing, laughing, et cetera, might be destroyed to some degree. Instead of making a purse string suture, I put in a series of sutures, one in the esophageal wall, one in the muscle wall and one in the mucosal wall of the trachea. That held for three days, and the same symptoms then developed to a mild degree; so there was definite indication of a leak.

I waited six weeks, and those were very long weeks, because I was anxious to cure the patient; then, on exposing the fistula again by the same procedure, I found that it was only 2 or 3 mm. in diameter; it was a simple procedure then to denude the tissues and to put a purse string suture around the fistula and tie it off. The child made an uneventful recovery. Does that answer your question?

DR. THOMAS CARMODY, Denver: Yes, thank you! I read it, but I thought it would be nice to explain it to the others.

DR. CHARLES J. IMPERATORI, New York: In answer to Dr. Iglauer's question as to whether the child had been given iodized poppyseed oil and barium sulfate, I suspect that barium sulfate had been given because at the time he was a few months old a diagnosis of pylorospasm had been made. He had been operated on for that, and also for cardiospasm.

I know Dr. Louis Clerf saw some of the roentgenograms. It was a definite cardiospasm, but none of this material ever entered the child's trachea, Dr. Iglauer; that is the strange thing. I know Dr. Clerf has another case just like this, with exactly the same lesions.

Barium may be placed into the esophagus in the attempt to make a roentgenogram; apparently, in these cases of congenital fistula, when the material reaches the fistula, the latter contracts and none of the medium gets through the fistula. It is only when the patient is in the prone position that there is regurgitation, and some of the material gets into the trachea and produces the syndrome of coughing, strangling, etc.

As to the stenosis in case 1, when I previously presented this case, I received the impression from the discussions that it was best to keep on dilating the stricture, and this I did.

DR. FREDERICK FIGI, Rochester, Minn.: I should think one might get a cure by laying open the larynx, inserting a skin graft, putting in the closure and inserting an arched cartilage bar if necessary.

DR. CHARLES J. IMPERATORI, New York: That was what I wanted to do, but I was told under no circumstances to do it. As I had never had any experience with the implantation of a cartilage arch, I was not certain it would hold, and for that reason I did not do it.

Penetrating Wound of the Neck with Unusual Complications and Sequelae.
DR. HENRY B. ORTON, Newark, N. J.

A white man aged 43 was first seen by Dr. Victor Knapp, of Asbury Park, N. J., on Nov. 17, 1941, with the complaint of hemoptysis since October 13. On October 6 he accidentally ran a splinter into the right side of his neck. He pulled it out and thought nothing more of the incident, although it was followed by dysphagia for about one month. Laryngoscopic examination showed fresh blood welling up into the trachea. Roentgenograms revealed pneumonitis but no evidence of tuberculosis or neoplasm. A small movable nodule developed in the right supraclavicular region. Later, glands in the right axilla became palpable. The question of a bronchogenic carcinoma was considered. In April 1942 the chest was opened in the third interspace. During manipulation of the upper lobe, a hemorrhage into the trachea occurred. This was controlled by placing a tourniquet around the upper lobe. The upper lobe was removed, and during the procedure a foreign body, which proved to be a splinter of wood, 2 inches (5 cm.) in length, was encountered, protruding into the thoracic cavity. This splinter had penetrated through the neck into the chest and into the apex of the lung, and in so doing it had either nicked the subclavian vein or eroded it from pressure.

DISCUSSION

DR. C. L. JACKSON, Philadelphia: Dr. Orton's case reminds me of one I had in which a penetrating wound was caused by shrapnel, with a piece of shrapnel lodging in the upper lobe of the left lung. A carcinoma developed in this lobe, and about two years ago a successful pneumonectomy was performed.

DR. HENRY B. ORTON, Newark, N. J.: I omitted reading part of my paper. When the lobe was sent to the laboratory, the pathologist, in sectioning it, said he found a carcinomatous gland along the region where the foreign body had penetrated.

Technic of Tracheotomy, with Use of the Sulfonamide Drugs in Prevention of Wound Infection. Dr. Gabriel Tucker, Philadelphia.

A tracheotomy wound is potentially an infected wound because of the fact that it communicates directly with the interior of the trachea. In the great majority of cases the trachea contains infection for which tracheotomy is required.

The methods of entering the trachea are two. First, when the trachea is to be preserved, the first ring is removed by subperichondrial elevation, and a cross incision is made in the trachea sufficient in length to accommodate the tracheotomy cannula. Second, vertical incision is made through three tracheal rings in the midline, the second, third and fourth preferably, the wound being of sufficient length to expose the region of the trachea selected for the opening and for the control of hemorrhage. The isthmus of the thyroid is cut if necessary. After the trachea is entered, the tracheotomy tube is inserted, and plain gauze impregnated with one of the sulfonamide drugs (preferably an aqueous solution of microcrystalline sulfathiazole) is packed around the tube. Plain gauze tape is wound around the tube and packed firmly against the trachea, so that it pushes aside the overlying tissues, including the muscles of the front of the neck. The usual split gauze dressing is then applied and the tapes are tied. This pack containing the sulfanilamide derivative accomplishes two things: It prevents subcutaneous emphysema, and the sulfonamide drug averts wound infection. The sulfonamide pack is used for the first few days and after that a smaller plain gauze pack; finally, no pack at all is required in the dressing of the wound.

The use of the technic of packing the wound with sulfonamide gauze has prevented wound infection and the development of subcutaneous emphysema and has allowed the wound to heal with minimal cicatrization and scarring of the neck.

DISCUSSION

Dr. Ralph A. Fenton, Portland, Ore.: My associates and I have been using this procedure for the past year and a half. At the University of Oregon Medical School we use an ordinary powder blower, which is much easier. We do not like sulfanilamide packed into the wound, because it is too soluble. We like sulfathiazole, but very little of it. We have found that by using the ordinary powder blower it is possible to get the same protective effect of "buttering" the cells with something which the germs do not like. The sulfonamide compounds produce not merely a bacteriostatic action, but one which really interferes with the attack of the organisms, particularly the staphylococci and streptococci, on these very vulnerable cells in the neck.

We have tried this treatment in a number of cases of tracheotomy in both children and adults. It is also in use at the Contagious Hospital, where tracheotomies are done in cases of diphtheria, and in all of these cases the perfectly insoluable sulfathiazole is found to work well.

Also, when there is a pulmonary complication, the powder can easily be blown in through the tube, but one must not use much of it.

Dr. Charles J. Imperatori, New York: I should like to amplify what Dr. Fenton said, particularly about blowing sulfathiazole into a wound. If too much is used it will accumulate and act like concrete, as it will not dissolve.

The term "buttering" which he used is a good one, and I suggest to Dr. Fenton that unless he has a very fine powder blower, a good method is that proposed last year by Dr. Gill, in which he takes an ordinary soda straw, cuts it in half, pushes it into the powder and with a little puff blows the powder in.

Dr. Thomas E. Carmody, Denver: In doing plastic work with cartilage grafts, I have administered sulfanilamide, as well as sulfathiazole, locally, closed the wound and had no trouble. I have also employed these drugs in operations on the mastoid and sinuses, experiencing no particular trouble from their use. As far as I know, this form of therapy was first used by Furstenberg. I have been employng it for about two years, but I have not used it in cases of tracheotomy. I see no reason that it should not be so employed.

The powder blower which Dr. Fenton suggests is good, and I think Dr. Gill's modification is also good. In the absence of a powder blower, drinking straws or tubes can be used. Anyway, the blowing of a little powder of one of the sulfonamide drugs into a wound is good therapy, but one must keep in mind that the sodium salt is irritating. If one gets too much into a wound, it will cake, as Dr. Imperatori suggests.

DR. GABRIEL TUCKER, Philadelphia: By packing tightly around the tube subcutaneous emphysema is prevented. By putting the pack in, by wrapping the tube down to the trachea, preventive medication is carried down to the trachea, and as the tube is removed the wound heals well. There is no difficulty about getting the tube in place if one packs around it in the way I demonstrated, leaving the retractors in.

In fact, in a number of cases other types of gauze were used, and there was always more or less infection, whereas with this method the hemorrhage is controlled and the wound allowed to heal. The infection is kept from extending into the dense tissues of the wound.

There is another point which might be of interest. The paper, a study on inhalation of microcrystals of sulfonamide drugs, which I mentioned and which is to be published, concerns itself with a special preparation of the sulfonamide compounds. The crystals are very small. I cannot give you the exact technic of how these crystals are produced, but the material is subjected to sound. It is done in one of the physiologic laboratories. An alkaline solution is treated, and the material comes out as a neutral salt of sulfathiazole. This has been used in sprays. It is white, looks much like cream and stays in suspension in physiologic solution of sodium chloride.

DR. GEORGE B. WOOD, Philadelphia: Is that suspension now available?

DR. GABRIEL TUCKER, Philadelphia: Smith, Kline and French Laboratories make some, which, I think, is only for experimental use.

Book Reviews

Diseases and Injuries of the Larynx. By Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., Honorary Professor of Broncho-Esophagology, Temple University, and Chevalier L. Jackson, A.B., M.D., M.Sc.(Med.), F.A.C.S., Professor of Broncho-Esophagology, Temple University, Philadelphia. Second Edition. Price $8. Pp. 633, with 221 pen, pencil and brush drawings, 11 colored plates and more than 100 halftones. New York: The Macmillan Company, 1942.

A second edition of "The Larynx and Its Diseases," by the Jacksons, has just come off the press. It retains in every detail the excellence of the original edition.

This volume totals 633 pages, of which 560 are devoted to the larynx and its diseases, and in these pages the reader will find the subject presented in the manner characteristic of the authors. Authority, interest, clarity, exactness and sound reasoning, aided by the numerous original sketches, schemas and colored drawings, as well as the 86 references cited, largely from the authors' own papers and compilations, though many are freely drawn from those of other distinguished laryngologists, all of these points, and many more, aid tremendously in presenting the teaching vividly and forcefully. By these means living pathology is literally demonstrated from the wealth of accumulated clinical data available, and the colored drawings, made by the authors themselves, are magnificently lifelike.

Functional values are particularly stressed, and the dysfunctions of the larynx are considered from every possible angle. The treatment for these conditions is outlined in meticulous detail. "Historical notes" append certain articles, not only enhancing the teaching value of the subject under discussion but indirectly establishing a basis for the truth in certain controversial problems.

Once again the Jacksons set forth one of the most powerful assets of their lifetime contribution to American laryngology, that is, the explanation of the intricate function of the airway along simple mechanistic principles.

The last chapter of the book takes up the subject of "War Surgery of the Larynx." Thirty-one pages are devoted to this phase, and the description is so vivid that the reader almost "sees" the effects of the cutting and stabbing weapons, the projectiles of small caliber and high velocity, the flame throwers and the incendiary bombs and the numerous wounds produced in the larynx by the different types of weapons described.

The endolaryngeal appearance is fully considered for every type of injury, and all treatment is outlined along broad principles with an exactitude that is almost proverbial.

The format, binding and print are a distinct improvement over the original edition.

This textbook is recommended to all otolaryngologists, bronchoesophagologists and graduate students in these specialties as a basic text to read, pencil-mark and return to again and again, and for general reference to all physicians and medical students.

The authors deserve high praise for this textbook and merit the plaudits of American laryngology for their numerous contributions to knowledge of the larynx and the tracheobronchial tree.